WADD TECHNICAL REPORT 60-634

EFFECT OF HEADWARD AND FORWARD ACCELERATIONS ON THE CARDIOVASCULAR SYSTEM

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JANUARY 1961

Contract No. AF 33(616)-5938

BIOMEDICAL LABORATORY
AEROSPACE MEDICAL LABORATORY
WRIGHT AIR DEVELOPMENT DIVISION
AIR RESEARCH AND DEVELOPMENT COMMAND
UNITED STATES AIR FORCE
WRIGHT-PATTERSON AIR FORCE BASE, OHIO

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FOREWORD

The study on which this report is based was accomplished in the cardiovascular and human centrifuge laboratory of the Mayo Foundation, Mayo Clinic, Rochester, Minnesota, under the direction of Dr. Earl H. Wood under Air Force Contract No. AF 33(616)-5938, Project No. 7220 and Task No. 71742. Dr. Edwin P. Hiatt, Biophysics Branch, Aerospace Medical Laboratory, Wright Air Development Division was the contract monitor. Doctor Wood was assisted in this study by Captains Evan F. Lindberg and Robert N. Headley, USAF (MC), both temporarily assigned to this project from the Acceleration Section of the Biophysics Branch, Aerospace Medical Laboratory, Wright Air Development Division, as well as by Dr. Hiram W. Marshall, Mr. William F. Sutterer and Dr. Terence F. McGuire of the Mayo Clinic. Work on this project started on 29 May 1958 and continued until 1 August 1960.

This study was made possible by the unstinting cooperation of many of our technical and professional colleagues in the Section of Engineering and Physiology, among which Miss Lucille Cronin, William Hoffman, Jim Isaacson, Ronnie Wilcox, Ed Tervo, and Mrs. Jean Frank are deserving of particular mention.

ABSTRACT

The purpose of this research was to measure cardiac output and related physiological variables in human subjects exposed to acceleration stress in various body orientations. Results of these experiments indicate that an average decrease in cardiac output of 22% of control values occurs in subjects exposed to headward accelerations of 4 g. No systematic change in cardiac output could be demonstrated when these same subjects were exposed to forward accelerations of up to 5 g. As the duration of these exposures to acceleration were increased to 10 minutes, no further alterations in output were demonstrable.

Exposure to headward acceleration caused decreases in right atrial and esophageal (intrathoracic) pressure and in the oxygen saturation of arterial blood which were proportional to the magnitude of the acceleration. Forward acceleration, however, caused relatively large increases in right atrial and esophageal pressure but a decrease in arterial oxygen saturation. The decrease in arterial oxygen saturation was prevented by breathing 99.6% oxygen.

PUBLICATION REVIEW

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LIST OF ILLUSTRATIONS

LIST OF TABLES

EFFECT OF HEADWARD AND FORWARD ACCELERATIONS ON THE CARDIOVASCULAR SYSTEM

The physiology of the visual blackout phenomenon encountered during headward acceleration as experienced in fighter aircraft of the World War II era, has been fairly well defined and reasonably effective preventative measures have been developed. Now the problems confronting the investigators in the field of acceleration physiology are related either to the much longer duration of exposures and on occasion to the much higher levels of acceleration which are anticipated in the flight of future aircraft and spacecraft. It is therefore important that the effect of acceleration lasting several minutes be known at magnitudes below that causing blackout, pain, unconsciousness, respiratory failure, and other gross endpoints. In order to determine the basis of decrements of human performance which may be attributed to failure of the cardiovascular system under these conditions, it is necessary to have measurements of cardiac output or some direct reflection of it under acceleration stress applied to human subjects in various vectors. Until recently no actual measurements of cardiac output during acceleration had been reported in human subjects. In September, 1959, Howard (ref. 8) reported the determination of cardiac output in two subjects using the direct Fick principle during exposure to headward acceleration over a period of approximately 5 minutes while reclining supine in the fully extended position. A 32 percent decrease in output at 2 g and a 40 percent decrease at 2.4 g were demonstrated.

The purpose of this investigation was to measure the cardiac output in human subjects during exposure to both headward (positive) and forward (transverse) acceleration while in the more usual seated position. The indicator-dilution technique with sudden single injections of dye into the right atrium and continuous recording of the resulting curves from arterial blood was used. These studies were carried out during 1 minute and 10 minute exposures to headward acceleration and during 10 minute exposures to forward acceleration. Since three separate series of experiments were conducted, the results of these studies are presented in three sections:

I. The Effect of Headward Acceleration of 1 Minute Duration

II. The Effect of Headward Acceleration of 10 Minutes Duration

III. The Effect of Forward Acceleration of 10 Minutes Duration

In general, the experimental methods employed in each series were nearly identical and are described in the next section. As the results from each series of experiments are presented, any necessary modifications of the procedures will be described.

This report is not intended to elucidate the entirety of the changes in cardiac output induced by acceleration, since all measurements of this parameter were made at intervals during a period of time from approximately 20 seconds to 10 minutes after the onset of acceleration. The initial compensatory cardiovascular response to the stress, therefore, had been completed

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(ref. 17) prior to the first determination. The results indicate that headward accelerations up to 4 g produce a decrease in cardiac output of varying degrees in the subjects studied. This decrease usually did not exceed 30 percent of the control value prior to centrifuge rotation, and no systematic change occurred as the duration of acceleration was prolonged to 10 minutes. On the other hand, forward accelerations of up to 5 g did not produce systematic changes in cardiac output over the 10 minute period during which determinations were carried out.

MATERIAL AND METHODS

Seven healthy physicians having previous experience on a human centrifuge served as subjects in these experiments. The vital statistics for these individuals are given in Table 1.

The intravascular catheters and radial artery needle used for the procedure were introduced while the subject rested supine on a fluoroscopic table equipped with an image intensifier assembly and prior to his entering the centrifuge cab. The venous catheter, which was a No. 5 Lehman type (length, 100 cm.; inside diameter, 0.8 mm.), was introduced by percutaneous needle puncture of a vein in the left forearm and advanced under fluoroscopic control so that its tip was located at the juncture of the superior vena cava with the right atrium.

The arterial catheter, which was a No. 4 F. (length: 60 cm.; inside diameter: 0.6 mm.), was introduced by a variation of the Seldinger technic following percutaneous needle puncture of the brachial artery near the left elbow. This catheter was advanced under fluoroscopic control so that its tip was in the subclavian artery just above the aortic arch. A roentgenogram of the chest was taken to verify the positions of both catheters (figure 1). The left radial artery was entered with a No. 20 gauge thin-walled needle (length: 8 cm.; inside diameter: 0.76 mm.).

The subject then walked to the centrifuge room and entered the cockpit, where the catheters and radial needle were connected to P23D Statham strain gauges mounted approximately at the level of the subject's third intercostal space at the sternum and positioned so that the sensing element was perpendicular (transverse) to the major vector of acceleration. A stopcock-controlled bypass system attached to these gauges permitted the frequent flushing of the catheters with heparinized Ringer's solution under pressure to prevent clotting (ref. 14). This same system was used for baseline checks and

Figure 1. Roentgenogram of the chest showing placement of the aortic and venous catheters.

Table 1

Vital Statistics of Experimental Subjects with Average Control (1 g) Values for Each Parameter Measured

Vital Statistics						Average Control Value						
Symbol	Subject	Age, yr.	Height, in.	Weight, kg.	Surface, sq. m.	Cardiac index L./min./m.^{2}	Heart rate, beats/min.	Stroke index ml./stroke/m.^{2}	Aortic pressure, mm. Hg.			Peripheral resistance, dynes sec. cm. $^{-5}$
									Syst.	Diast.	Mean	
△	1	29	74	72	1.98	5.1	78	65	118	73	88	760
	2	47	70	87	2.05	4.5	98	45	135	83	101	920
×	3	27	73	92	2.16	2.9	77	38	118	83	95	1230
○	4	35	71	77	1.96	3.3	77	43	145	94	111	1330
●	5	31	73	79	2.04	3.8	85	45	111	76	87	900
▲	6	30	72	75	1.95	2.6	72	36	133	86	102	1590
	7	34	68	55	1.70	4	85	52	118	83	95	1050

calibration of the manometer systems against known pressures (ref. 14) at intervals between the exposures to acceleration. A cuvette oximeter was interposed between the radial artery needle and its respective strain-gauge manometer. A two-way stopcock was used for immediately interchangeable connection of the radial artery cuvette system to the strain gauge for recording of pressure or to a mechanically controlled 30-ml. siliconed hypodermic syringe for withdrawal or infusion of blood at a constant rate of 25 ml. per minute.* The plunger of this syringe was mechanically coupled to a linear potentiometer for continuous recording of its position, and hence recorded the rate and volume of blood being withdrawn or reinfused into the artery.

Another two-way stopcock was interposed between the venous catheter and its strain gauge for immediately interchangeable connection of the catheter to that gauge for pressure recording or to a solenoid-controlled, pneumatically activated 8.5-ml. stainless steel syringe (ref. 5) filled with an aqueous solution of indocyanine green (Cardio-green) dye** (5 mg./ml.). The piston of this syringe was mechanically coupled to a linear potentiometer for continuous recording of its position, and hence of the time, duration, and volume of the sudden single injections of indocyanine green. The onset of injection was controlled manually by a push-button switch, and the duration (volume) was automatically held constant by an electrical holding circuit. Each injection of indocyanine green dye (ref. 2) consisted of 5 mg. of dye in a volume of 1 ml. The duration of each injection was 0.16 seconds. At the beginning and end of each experiment, 60 ml. samples of blood were withdrawn from the radial artery into syringes containing heparin. These samples were divided into aliquots to which known amounts of indocyanine green were added for calibration of the cuvette oximeter as described previously (ref. 4). Twenty ml. samples of both radial artery and superior vena cava blood were simultaneously withdrawn for Van Slyke analysis of oxygen content. Aliquots of these samples were reinfused through the cuvette oximeter as a check on the calibration of this instrument for blood oxygen saturation. The physical dimensions, dynamic response, and electrical circuitry used with this oximeter as well as the methods of calibration have been described previously (ref. 3, 4, 18). Ten seconds prior to each injection of the indicator, a constant-rate withdrawal of blood from the radial artery through the cuvette oximeter at 25 ml. per minute was begun, and it was continued for approximately 30 seconds after the injection. This provided a continuous record of the dye concentration in arterial blood during the initial circulation of the indicator. The blood withdrawn during the dilution curve was reinfused into the radial artery upon completion of each dye curve. Cardiac output was calculated from each of these dilution curves by use of the method outlined by Stewart, Hamilton, and others (ref. 9).

Respirations were recorded by a thermocouple as temperature variations in an oral airway, the nostrils being occluded so as to necessitate breathing through the mouth. The electrocardiogram was recorded from leads taped to the chest in a modified lead II position. The subject's reaction

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* This mechanically controlled syringe assembly was obtained from the Harvard Apparatus Co., Dover, Mass.

** Supplied through the courtesy of Hynson, Westcott, and Dunning, Inc.

times to a heterogeneous series of red light signals mounted at his fixation point (105 cm. from the eyes) and blue-green light signals mounted bilaterally in this same vertical plane on a horizontal line 16° from his fixation point were recorded throughout each exposure to headward acceleration and during the control determinations of cardiac output. The rate of rotation of the centrifuge was recorded continuously by means of a tachometer, and also the acceleration in the centrifuge cockpit was recorded from an accelerometer mounted at the level of the subject's heart. In part of the experiments the angle of the centrifuge cab from the vertical was also recorded by means of a potentiometer mechanically coupled to the axle on which it rotated. These variables and the aortic pressure were recorded continuously during each exposure. The right atrial pressure was also recorded, except during the instant of the dye injection, and likewise the radial artery pressure except for the periods of withdrawal of blood for recording of the dilution curves.

Upon completion of the series of centrifuge accelerations and without alteration of the position of the subject or strain gagues, fluid-filled thistle tubes were attached to the subject in the mid-axillary line on both sides of the thorax, and the menisci of the fluid columns in these two tubes adjusted to the midpoint of the maximum antero-posterior chest dimension at the level of the third intercostal space at the sternum. Each of the strain gauges was closed off from the pressure being recorded and opened instead to the fluid column in this thistle tube system. Through individual 5-second exposures made at each level of acceleration studied, the shift in reference level (mid chest at the level of the third intercostal space at the sternum) during acceleration was recorded as the subject was pushed down into the seat and the manometer systems were subjected to the levels of acceleration at which the physiologic recordings were made. The necessary corrections for these shifts in base line were included in the measurement of all pressures recorded during acceleration.

The information gathered by the instruments on the centrifuge was transmitted via a mercury trough commutator system to galvanometers in a separate recording room. By means of a recording assembly described elsewhere (ref. 15), photokymographic records of these galvanometer tracings were taken on two cameras simultaneously at paper speeds of 1.25 or 5 and 25 or 150 mm./sec.

During the two series of headward acceleration studies, activation of the automatic blood sampling and dye injection syringes as well as the turning of stopcocks for blood withdrawal, injection of indicator, and flushing of the catheter assemblies were performed by an observer lying prone above the subject on top of the centrifuge cockpit (figure 2). The stopping and starting of the centrifuge and the light signals

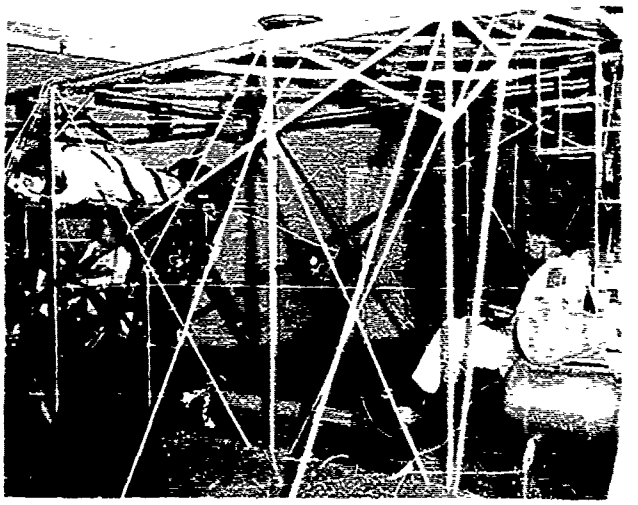

Figure 2. Cockpit end of human centrifuge showing subject's position in relation to observer lying prone on top of cockpit and to observer sitting near center of rotation.

to the subject were under the control of a second observer positioned near the center of rotation of the centrifuge so as to have a clear view of the cockpit and the subject and first observer.

From the beginning of rotation the centrifuge required about 5 seconds to attain a speed of 15 revolutions per minute which was equivalent to a headward vector of acceleration of 1.5 g. The rate of onset of acceleration was then maintained at about 1 g per second until the plateau level of acceleration was attained. The distance from the center of rotation to the subject's heart level was 15 feet. For all the exposures to headward acceleration the subject's feet were supported in the usual cockpit position with heels 32 cm. below the buttocks. The backrest of the seat was inclined backwards 13° from the vertical and the subject's occiput was in contact with a headrest so that the head and neck were afforded minimal support in an approximately vertical position. The subject faced in the direction of rotation, which was counterclockwise as viewed from above the centrifuge. The centrifuge room was in semi-darkness during the exposure, the cockpit and its occupants being illuminated by a shaded 200 watt bulb mounted 10 feet from and directed towards the cockpit.

RESULTS

I. The Effect of Headward Acceleration of One Minute Duration

For this series of experiments, 6 subjects underwent a total of 51 separate exposures to headward acceleration of 60 seconds duration at plateau acceleration levels which ranged from 2 to 4 g. There were 17 exposures to 2 g, 22 exposures to 3 g, and 12 exposures to 4 g. A dye-dilution curve was recorded during each of these exposures. Thirty-five control (1 g) determinations of cardiac output were interspersed with the 51 determinations carried out during centrifuge rotation. The subjects wore a cutaway type (g-3A) anti-g suit which was inflated via a g-activated valve to a pressure of approximately 200 mm. Hg. during one of the exposures to each of the three levels of headward acceleration studied. In 4 of the 6 subjects, a plastic catheter was inserted into the esophagus via a nostril so that its tip was at the level of the mid cardiac silhouette. This catheter was attached to a P23D Statham strain-gauge manometer which provided a continuous record of intraesophageal (intrathoracic) pressure.

A typical photokymographic recording obtained during a 60 second exposure to a headward acceleration of 3 g without inflation of the g-3A suit is shown in figure 3. Note that the injection of the indicator for measurement of cardiac output was not made until after the initial 10-second period of cardiovascular insufficiency induced by the acceleration. The sequence of an initial period of failure followed by cardiovascular compensation induced by headward acceleration has been described previously (ref. 17); and although it was evident in all of the exposures carried out in this series of experiments, as illustrated in figure 3, it will not be further detailed in this presentation. It should be remembered and emphasized, however, that all of this section's data as to changes in cardiac output

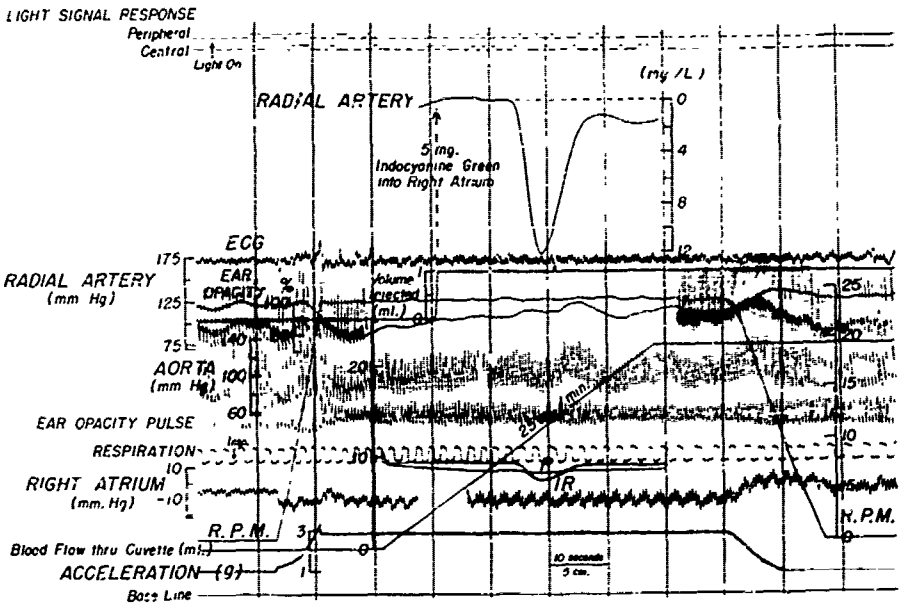

Figure 3. Typical photokymographic record of indicator-dilution curve and other variables during exposure of healthy man to headward acceleration of 3 g for 70 seconds.

Note: (1) the initial decrease and subsequent compensatory rise in arterial pressure recorded in both the aorta and radial artery which occurred within the first 15 seconds of the exposure to acceleration; (2) a downward deflection in ear opacity with the onset of acceleration, denoting a decrease in blood content of the ear, evidently correlated with the variations in arterial pressure; (3) the initial decrease in the amplitude of the ear opacity pulse indicating a temporary fall in arterial pressure at ear level; (4) the cessation of the radial artery pressure tracing when the radial needle was opened to the automatic blood withdrawal syringe and flow started through the cuvette oximeter; (5) the interruption of the right atrial pressure tracing as the catheter was opened momentarily to the automatic injection syringe for injection of dye; (6) the rapid injection of 1 ml. of the dye solution (5 mg.) made 20 seconds after the onset of acceleration; (7) the large deflection in the recording of the concentration of indocyanine dye in radial artery blood associated with the initial traversal of the dye blood mixture through the central circulation followed by a much smaller deflection denoting the recirculation of the mixture; (8) the decrease in transmission of red light in arterial blood before injection of the indicator, signifying a drop in arterial blood oxygen saturation of approximately 8 per cent during the first 30 seconds of the exposure, which occurred without evident abnormality in the rate or depth of respiration.

The blood withdrawn for recording of the dilution curve was reinfused into the radial artery shortly after termination of the exposure.

induced by headward acceleration were obtained during periods extending approximately from 20 to 40 seconds after the attainment of the plateau level of acceleration. These data, therefore, pertain only to this particular period of cardiovascular compensation that occurs after the rapid onset of exposure to a plateau level of headward acceleration. They are not pertinent to the much more dramatic cardiovascular alterations that occur during the initial 5 to 15 seconds of such exposures.

The results of these experiments are shown in figures 4 through 8, which depict alterations in cardiac output, heart rate, stroke volume, mean aortic pressure* and total peripheral resistance ** during accelerations with and without inflation of the anti-g suit. The measurements of heart rate and mean aortic pressure were made from that portion of the record from which the calculations of the cardiac output and stroke volume were made. Each subject was assigned a number and every determination of each variable was assigned a letter in alphabetic sequence. Thus the temporal sequence of determinations can be followed by means of the alphabetic designations, and the individual variability in the results can be visualized.

The average level and range of the control (1 g) values for the cardiac indexes of these subjects, seated in the cockpit were 3.6 and 2.5 to 5.9 liters per minute per square meter of body surface. The values were closely similar to the corresponding values of 3.5 and 2.5 to 4.4 L./min./m^2 obtained in this laboratory from healthy subjects resting supine on a padded table (ref. 1). During the course of each experiment, the control values for cardiac output in individual subjects ranged from ± 10 to ± 20 percent of the average control value (left panel, figure 4). That this variability was related in part to some degree of anxiety during the procedures is suggested by the fact that the level of cardiac output for the first determinations at 1 g (values designated by letter A) and the level for the second determinations (letter B), which were made at 2 g, tended to be significantly higher than those recorded later, when presumably the subject's initial level of anxiety had been dispelled by the successful initial experience of centrifuge rotation under these circumstances. With exclusion of this first exposure to acceleration, which was invariably at the 2 g level, there was a decrease in cardiac output during exposure to acceleration in each instance (left panel, figure 4). When all values obtained during acceleration are included and compared to the temporally contiguous control values, the average decreases in cardiac output were 7, 18, 22 percent at 2, 3, and 4 g, respectively (table 2).

When the skeleton type of anti-g suit was inflated to 200 mm. Hg. during exposure to acceleration, the average values obtained for cardiac output were slightly greater than the values obtained without the protection afforded by the nonuniform and incomplete degree of pressurization of the lower part of the body that is provided by this relatively ineffective anti-blackout suit. The average differences from the values without suit inflation did not, however, attain statistical significance (figure 4, table 2).

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* Mean aortic pressure: diastolic pressure + 1/3 pulse pressure.

** Total peripheral resistance: $\frac{\text{mean aortic pressure (mm. Hg.)} \times 79920}{\text{cardiac output (L./min.)}}$

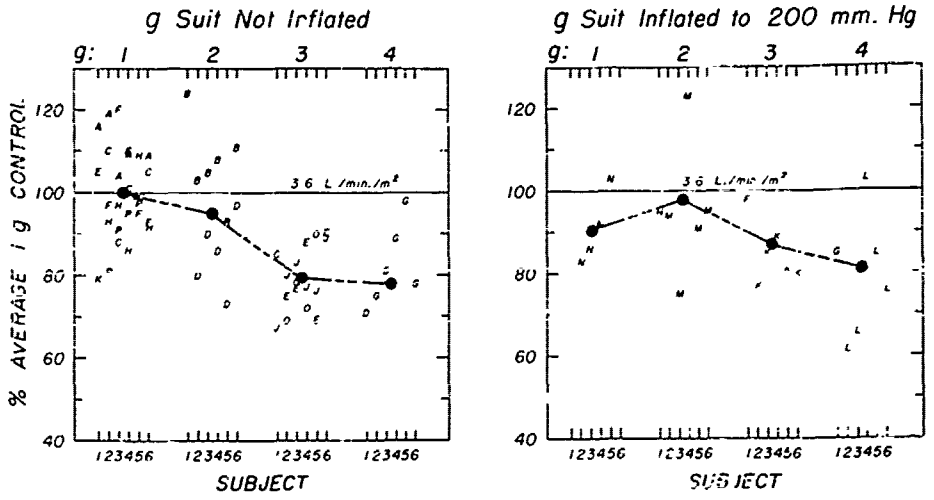

Figure 4. Effect of headward acceleration on cardiac output.

Values were determined from dye-dilution curves recorded at the radial artery in six subjects 20 to 40 seconds after the attainment of the plateau levels of acceleration indicated on the abscissa. Values for the individual subjects are plotted in a vertical line over the appropriate numerals 1 to 6, shown on the abscissa at each of the four levels of acceleration studied. The temporal sequence of individual determinations in each experiment on the six subjects is indicated by plotting the values in alphabetical sequence from A up to, for example, P -- that is, from determinations 1 up to 15 -- the maximal number of dye-dilution curves carried out in any of the subjects. The letter I was omitted to avoid confusion with the numeral 1.

Each value for each subject is plotted as percentage of the average cardiac output obtained in the three to five determinations made from him in the control situation (seated in the centrifuge cockpit with the centrifuge stationary, hence exposed only to the 1 g acceleration of gravity). The averages of the control values obtained from each subject are given in table 1. This makes possible the conversion of the percentage values shown in figures 3 through 7 to absolute values should the reader desire.

Note that the initial two determinations (A and B) tended to give the highest values, apparently as the result of some degree of apprehension which was dispelled to an appreciable degree by the successful completion of the first period of centrifuge rotation under the unusual conditions pertaining in this experiment. Despite the ±20 per cent variation in the control values for cardiac output, a significant systematic decrease in cardiac output during this period of exposure to a plateau level of acceleration is evident. This effect was not systematically altered by inflation of a type g-3A antiblackout suit during the period of exposure (right panel).

Table 2

Average Decreases in Cardiac Index From Temporally Contiguous Control Values Which Occurred in Subjects 20 to 40 Seconds After the Onset of Exposures to Plateau Levels of Headward Acceleration With and Without Inflation of the G-3A Suit

Subjects	G-suit not inflated				Subjects	Uninflated		G-suit inflated	
	Control (1 g) value, L./min./m.^{2}	Acceleration	Decrease during acceleration			Control (1 g) value, L./min./m.^{2}	Acceleration	Decrease during acceleration	
			L./min./m.^{2}	Per cent				L./min./m.^{2}	Per cent
-	-	-	-	-	4	(4) 3.28 [2.64 - 3.73]	1 g	(4) -0.01• [-.19 to .14]•	(4) 0 [-5 to 5]•
6	(11) 3.74 [2.55 - 5.90]	2 g	(11) 0.31 [-.10 to 1.32]•	(11) 7 [-7 to 27]•	6	(6) 3.27 [2.46 - 4.00]	2 g	(6) -0.28• [-.92 to .46]•	(6) -7• [-29 to 17]•
6	(16) 3.58 [2.46 - 5.90]	3 g	(16) 0.67 [-.10 - 1.60]	(16) 18 [4 - 35]	6	(6) 3.30 [2.46 - 5.35]	3 g	(6) 0.45 [-.10 to 1.08]•	(6) 12 [-3 to 26]•
6	(11) 3.72 [2.46 - 5.35]	4 g	(6) 0.88 [.17 - 1.90]	(6) 22 [4 -45]	6	(6) 3.30 [2.46 - 5.35]	4 g	(6) 0.73 [-.56 to 1.41]•	(6) 18 [-19 to 34]•

( ) Number of individual determinations.
[ ] Range of individual determinations.
• Minus sign indicates an increase during acceleration.

The values of 82 and 69 to 101 beats/min. for the average and range of control (1 g) heart rates recorded when the subjects were seated in the cockpit (figure 5) were similar to the corresponding values of 73 and 59 to 113 beats per minute obtained in this laboratory for healthy subjects undergoing cardiac catheterization while resting supine (ref. 1). The heart rate increased during acceleration as expected (figure 5). The average increases,

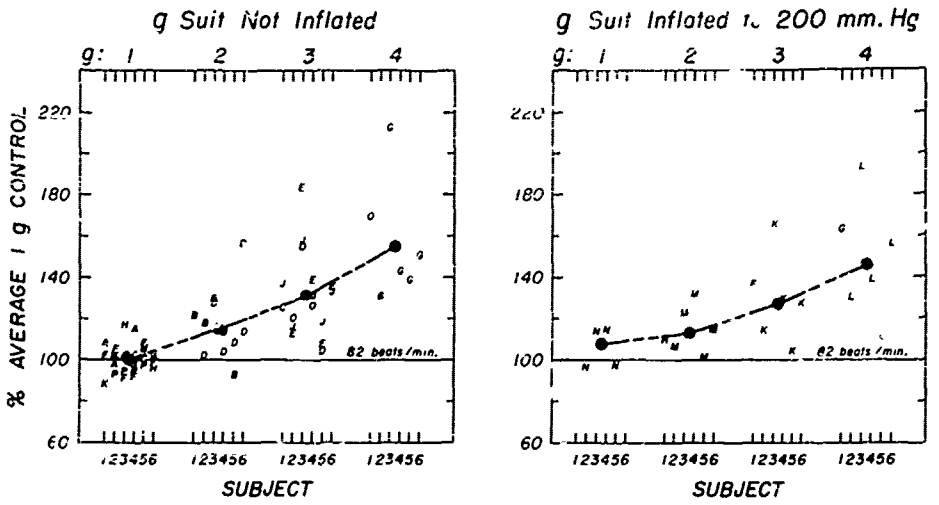

Figure 5. Effect of headward acceleration on heart rate of six healthy subjects.

The values plotted were obtained during a period extending from approximately 20 to 40 seconds after the attainment of the plateau level of acceleration. (See legend 3 for explanation of symbols used.

as compared with the temporally contiguous control (1 g) values, were 14, 35, and 56 per cent at 2, 3, and 4 g, respectively (table 3).

The average increases in heart rate during exposures with the g-3A suit inflated to 200 mm. Hg. were slightly less than the values without suit inflation, but the differences did not attain statistical significance (figure 5, table 3).

Values for stroke index were calculated for each determination of cardiac output by dividing the cardiac index value by the average heart rate measured during the period of inscription of the dilution curve (figure 6). As would be expected from the previous comparisons, the values of 44 and 31 to 71 ml./stroke/m^2 for average and range during the control period were closely similar to the 46 and 37 to 63 ml./stroke/m^2 obtained from the group of subjects catheterized in the supine position (ref. 1). As compared with the temporally contiguous control values (table 4), the stroke index decreased by 24, 37, and 49 percent at 2, 3, and 4 g, respectively. Although the average decrease in stroke index was less during acceleration with the g-3A suit inflated, the average differences were not statistically significant.

Table 3

Average Increases in Heart Rate From Temporally Contiguous Control Values Which Occurred in Subjects 20 to 40 Seconds After the Onset of Exposures to Plateau Levels of Headward Acceleration With and Without Inflation of the G-3A Suit

Subjects	G-Suit not inflated				Subjects	Uninflated	G-Suit Inflated		
	Control (1 g) value, beats/min.	Acceleration	Increase during acceleration			Control (1 g) value, beats/min.	Acceleration	Increase during acceleration	
			Beats/min.	Per Cent				Beats/min.	Per Cent
-	-	-	-	-	4	(4) 79 [72 - 90]	1 g	(4) 9 [0 - 16]	(4) 12 [0 - 22]
6	(11) 84 [72 - 100]	2 g	(11) 11 [-4 to 40]	(11) 14 [-5 to 55]*	6	(6) 76 [69 - 90]	2 g	(6) 17 [4 - 29]	(6) 23 [5 - 40]
6	(16) 80 [69 - 101]	3 g	(16) 27 [14 - 71]	(16) 35 [14 - 61]	6	(6) 83 [71 - 100]	3 g	(6) 20 [1 - 42]	(6) 26 [1 - 49]
6	(11) 82 [71 - 101]	4 g	(6) 45 [28 - 86]	(6) 56 [27 - 110]	6	(6) 83 [71 - 100]	4 g	(6) 37 [7 - 64]	(6) 45 [8 - 74]

( ) Number of individual determinations.
[ ] Range of individual determinations.
* Minus sign indicates a decrease during acceleration.

Table 4

Average Decreases in Stroke Index From Temporally Contiguous Control Values Which Occurred in Subjects 20 to 40 Seconds After the Onset of Exposures to Plateau Levels of Headward Acceleration With and Without Inflation of the G-3A Suit

G-suit not inflated					G-suit inflated				
Subjects	Control (1 g) value, ml./stroke/m.^{2}	Acceleration	Decrease during acceleration		Subjects	Uninflated Control (1 g) value, ml./stroke/m.^{2}	Acceleration	Decrease during acceleration	
			Ml./stroke/m.^{2}	Per Cent				Ml./stroke/m.^{2}	Per cent
-	-	-	-	-	4	(4) 41 [34 - 58]	1 g	(4) 6 [1 - 9]	(4) 14 [1 - 24]
6	(11) 45 [32 - 71]	2 g	(11) 10 [1 - 27]	(11) 24 [2 - 54]	6	(6) 43 [34 - 58]	2 g	(6) 4 [-3 to 14]*	(6) 9 [-7 to 30]*
6	(16) 44 [31 - 72]	3 g	(16) 17 [6 - 31]	(16) 37 [13 - 49]	6	(6) 48 [33 - 67]	3 g	(6) 14 [9 - 20]	(6) 30 [23 - 42]
6	(11) 43 [33 - 67]	4 g	(6) 23 [12 - 40]	(6) 49 [27 -65]	6	(6) 43 [33 - 67]	4 g	(6) 18 [7 - 34]	(6) 43 [18 - 64]

( ) Number of individual determinations.
[ ] Range of individual determinations.
* Minus sign indicates an increase during acceleration.

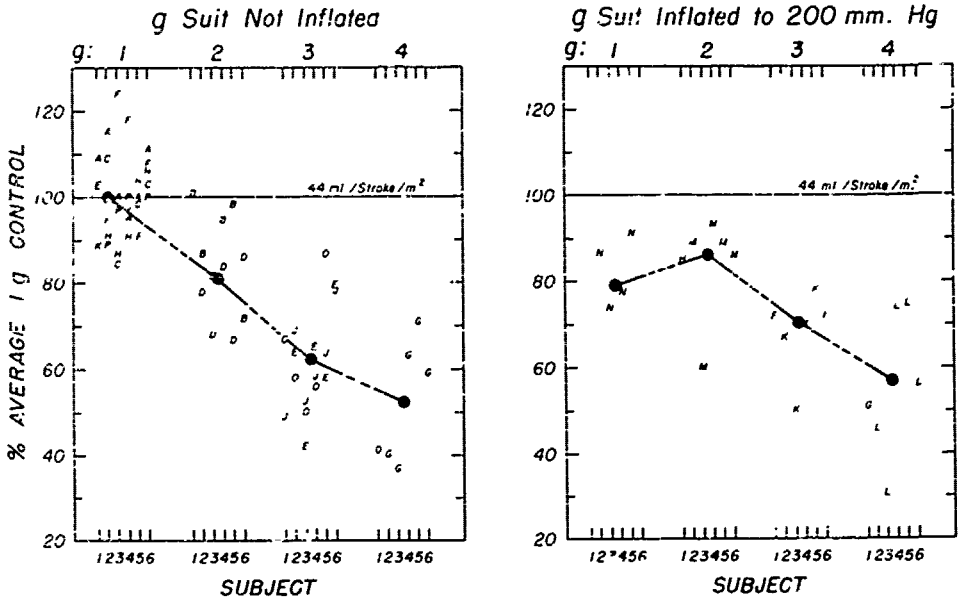

Figure 6. Effect of headward acceleration on stroke volume of six healthy subjects.

The values were obtained during a period extending from approximately 20 to 40 seconds after the attainment of the plateau level of acceleration. (See legend 3 for explanation of symbols used.)

The values of 97 and 74 to 113 mm. Hg. for the average and range of mean aortic pressure obtained during the control (1 g) period in this group of subjects were similar to the levels of mean arterial pressure obtained in this laboratory in resting healthy subjects (ref. 1). During exposure to acceleration without inflation of the antiblackout suit, the aortic pressure at heart level decreased slightly in the initial 5 to 8 seconds of the exposure at the plateau level of acceleration. Aortic pressure then increased as the cardiovascular compensatory reactions became effective, so that during the period from 20 to 40 seconds after the beginning of the exposure the mean aortic pressure averaged 9, 21, and 27 percent greater at 2, 3, and 4 g, respectively, than the temporally contiguous control level of pressure (figure 7, table 5).

Inflation of this rather ineffective antiblackout suit to 200 mm. Hg. in the control situation produced a slight increase - averaging 10 mm. Hg. - in aortic pressure (right panel, figure 7, table 5). Inflation of this suit at the beginning of exposure to acceleration prevented the initial slight decrease in aortic pressure at heart level at the onset of the exposure, although not to the extent of producing a striking degree of hypertension at heart level, such as has been reported previously, for other highly effective models of antiblackout suits (16). The slight increase in aortic pressure produced by

Table 5

Average Increases in Mean Aortic Pressure From Temporally Contiguous Control Values which Occurred in Subjects 20 to 40 Seconds After the Onset of Exposures to Plateau Levels of Headward Acceleration With and Without Inflation of the G-3A Suit

	G-suit not inflated					Uninflated	G-suit inflated		
Subjects	Control (1 g) value, mm. Hg.	Acceleration	Increase during acceleration		Subjects	Control (1 g) value, mm. Hg.	Acceleration	Increase during acceleration	
			Mm. Hg.	Per cent				Mm. Hg.	Per Cent
-	-	-	-	-	4	(4) 96 [89 - 103]	1 g	(4) 10 [0 - 19]	(4) 10 [0 - 19]
6	(11) 98 [83 - 112]	2g	(11) 9 [0 - 17]	(11) 9 [0 - 17]	5	(5) 95 [89 - 103]	2 g	(5) 24 [12 - 32]	(5) 25 [13 - 31]
6	(16) 95 [89 - 113]	3 g	(16) 20 [4 - 31]	(16) 21 [3 - 42]	6	(6) 96 [74 - 105]	3 g	(6) 34 [24 - 38]	(6) 36 [23 - 43]
6	(11) 99 [74 - 105]	4 g	(6) 26 [14 - 33]	(6) 27 [13 - 39]	5	(5) 93 [74 - 105]	4 g	(5) 40 [27 - 54]	(5) 44 [26 - 54]

( ) Number of individual determinations.
[ ] Range of individual determinations.

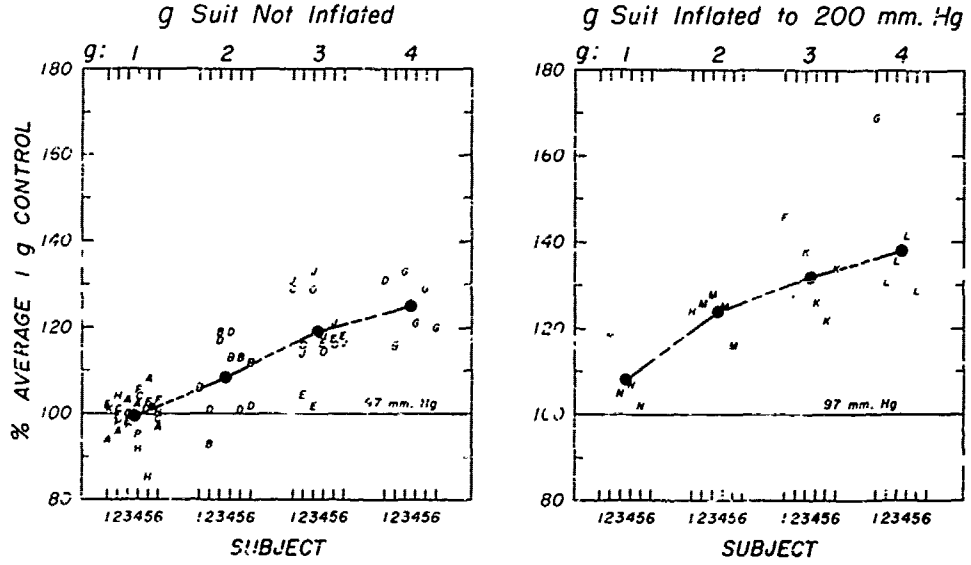

Figure 7. Effect of headward acceleration on mean aortic pressure at heart level (third intercostal space at the sternum) in 6 healthy subjects.

The values plotted were obtained during the period of recording of an indicator-dilution curve 20 to 40 seconds after the attainment of the plateau level of acceleration. (See legend 3 for explanation of symbols used.)

Note (right panel) that the aortic pressure when the type g-3A antiblackout suit was inflated at 1 g was consistently higher than the average control pressure. During exposures with the suit inflated pressures were significantly higher than in the temporally contiguous exposures without lower-body pressurization (table 5).

inflation of the suit was maintained during the period from 20 to 40 seconds after the beginning of the exposure, so that the aortic pressure during this period averaged approximately 15 mm. Hg. higher than without pressurization of the lower body. This increase in arterial pressure produced by the suit, particularly during the initial seconds of the exposure, is undoubtedly the basis for the moderate degree of protection against the occurrence of visual symptoms which this suit afforded. In these six subjects the average protection afforded by inflation of the suit against the development of visual symptoms was approximately 1 g.

The average and range of control (1 g) values of 1120 and 560 to 1750 dyne sec. cm.$^{-5}$ for systemic arterial pressure/flow ratio (systemic vascular resistance) in this group of subjects were closely similar to the corresponding values of 1130 and 745 to 1750 dyne sec. cm.$^{-5}$ obtained previously in this laboratory from healthy subjects (ref. 1). Exposure to acceleration was associated uniformly with an increase in the systemic

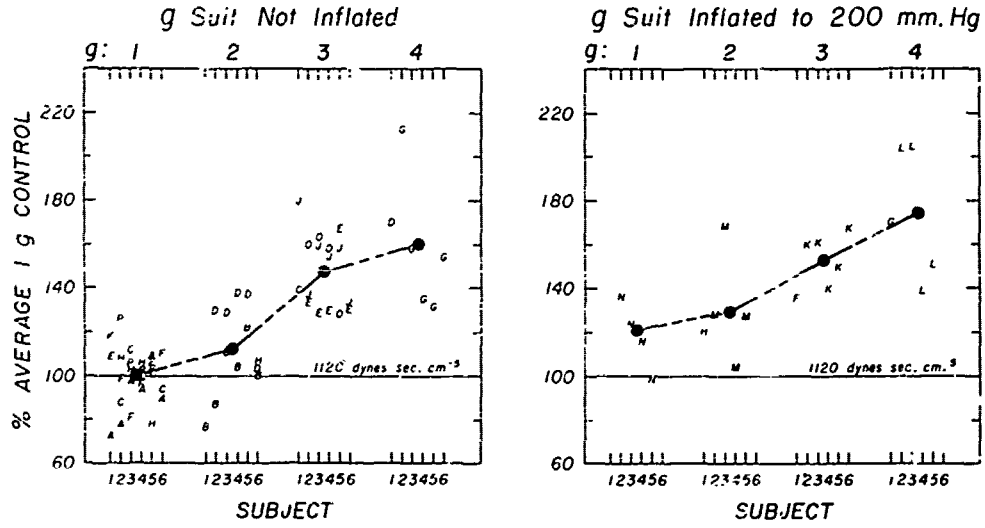

Figure 8. Effect of headward acceleration on systemic arterial pressure/flow ratios (systemic vascular resistance) of six healthy subjects.

The values were obtained during a period extending from approximately 20 to 40 seconds from the attainment of the plateau level of acceleration. Note that -- in contrast to the cardiac output (fig. 3) -- during the initial two determinations (A and B) the level of peripheral vascular resistance tended to be lower than for subsequent determinations. (See legend 3 for explanation of symbols used).

pressure/flow ratio as viewed from heart level (figure 8, table 6). The increase in this ratio averaged 17, 41, and 59 percent at 2, 3, and 4 g, respectively. The magnitude of the increase in "peripheral vascular resistance" induced by acceleration tended to be somewhat greater when the antiblackout suit was inflated at the onset of the exposure, but this difference from the unprotected exposures did not attain statistical significance (table 6).

The oxygen saturation of arterial blood as determined by cuvette oximetry from radial artery blood began to decrease within 10 seconds after the onset of the plateau level of acceleration (figure 3). The average and range of the minimum values for arterial blood oxygen saturation attained before inscription of the dye-dilution curve shown in table 7, revealed a systematic decrease within the first 15 seconds of an exposure to 4 g headward acceleration. With inflation of the anti-g suit to 200 mm. Hg. pressure, this same degree of desaturation was reached at the 3 g level. Saturation values obtained with the anti-g suit inflated in this manner were consistently lower than those recorded with the suit uninflated at each level of acceleration studied. Injection of the dye and the resulting dilution curve recorded by the infra-red photocell of the cuvette prevented any further determination of blood oxygen saturation during the remainder of the exposures when cardiac output studies were being carried out.

Table 6

Average Increases in Systemic Arterial Pressure/Flow Ratios (Systemic Vascular Resistance) From Temporally Contiguous Control Values in Subjects 20 to 40 Seconds After the Onset of Exposures to Plateau Levels of Headward Acceleration With and Without Inflation of the G-3A Suit

G-suit not inflated					G-suit inflated				
			Increase during acceleration					Increase during acceleration	
Subjects	Control (1 g) value, dynes sec. cm.$^{-5}$	Acceleration	Dynes sec. cm.$^{-5}$	Per cent	Subjects	Control (1 g) value, dynes sec. cm.$^{-5}$	Acceleration	Dynes sec. cm.$^{-5}$	Per cent
-	-	-	-	-	4	(4) 1190 [940 - 1370]	1 g	(4) 110 [-50 to 220] *	(4) 8 [-5 to 17] *
6	(11) 1100 [560 - 1490]	2 g	(11) 191 [30 - 490]	(11) 17 [4 - 47]	5	(5) 1135 [900 - 1370]	2 g	(5) 204 [20 - 760]	(5) 17 [1 - 58]
6	(16) 1170 [560 - 1750]	3 g	(16) 462 [210 - 740]	(16) 41 [19 - 103]	6	(6) 1150 [700 - 1700]	3 g	(6) 590 [260 - 970]	(6) 53 [34 - 92]
6	(11) 1160 [700 - 1750]	4 g	(6) 634 [350 - 1000]	(6) 59 [34 - 105]	5	(5) 1040 [700 - 1410]	4 g	(5) 744 [440 - 1260]	(5) 74 [31 - 100]

( ) Number of individual determinations.
[ ] Range of individual determinations.
* Minus sign indicates a decrease during acceleration.

Table 7

Minimum Values for Arterial Blood Oxygen Saturation* Attained
During the first 15 Seconds of Exposure to Plateau Levels of Headward Acceleration

(average values - 5 subjects)

	Control Value (% Saturation)	Value During Exposure to Acceleration (% Saturation)		
	1 g	2 g	3 g	4 g
g-suit not inflated	(24) 97 [96 - 99]	(10) 96.5 [95.5 - 97.5]	(14) 94.5 [92 - 98.5]	(5) 93 [91 - 96]
g-suit inflated to 200 mm. Hg.	(4) 96 [95.5 - 97]	(5) 94 [92 - 96]	(5) 93 [90 - 96]	(4) 89 [87 - 91.5]

( ) Number of individual determinations
* Determined by cuvette oximetry from blood being withdrawn from the radial artery
[ ] Range of individual determinations

II. The Effects of Headward Acceleration of 10 Minutes Duration

The same experimental methods used in the first series of headward acceleration studies were utilized in this series. With the onset of the plateau level of acceleration the first injection of dye was made within 20 to 30 seconds. As soon as this dilution curve was completed and while acceleration continued, the automatic withdrawal syringe was activated to reinfuse the blood withdrawn during inscription of the curve. Upon completion of the reinfusion of blood into the radial artery, withdrawal was again begun and dye injected for the second determination of cardiac output. This procedure was repeated as often as 5 times during the 10 minute exposure to acceleration, each withdrawal and infusion requiring approximately 100 to 120 seconds.

Each of 5 subjects was exposed to 3 separate plateau levels of acceleration of up to 10 minutes duration ranging from 2 to 3½ g. A total of 25, 24, and 18 dye dilution curves were recorded during the exposures to 2, 3, and 3½ g respectively. Twenty-nine control (1 g) determinations of cardiac output were performed prior to and following the 67 determinations carried out during centrifuge rotation. In addition to these controls, the cardiac outputs of 4 of the 5 subjects were determined 5 separate times in rapid succession during a 10 minute period with the centrifuge stationary.

The results of these studies are shown in tables 8 through 12 which depict alterations in cardiac output, heart rate, stroke volume, mean arterial pressure, and total peripheral resistance during prolonged exposures to headward acceleration. As in the previous section, the measurement of heart rate and mean aortic pressure were made from that portion of the record from which calculations of the cardiac output were made.

The average and range of control values for cardiac output (table 8) are comparable to those in table 2 in the previous series of experiments. It should be noted that on the average the cardiac output varied plus or minus 11 percent in the control condition over a 10 minute period of time. The range of individual variation was -20 to +22 percent of the individual's average control value. With the onset of acceleration there was a decrease in cardiac output on the average during the first minute of exposure to all three levels of acceleration studied. As the exposure continued for 10 minutes, multiple determinations of cardiac output, at approximately 2 minute intervals, failed to demonstrate any systematic decrease or increase in the average values, and the variation in these values did not exceed that of the control period. The average decrease in cardiac output ranged from 5 to 17, from 6 to 20, and from 7 to 28 percent of the individual's control value prior to exposure to 2, 3, and 3½ g, respectively. A graphic representation of the individual variations in cardiac output during the 10 minute exposure to 3 g preceded by the 5 control determinations in rapid succession are shown in figure 9. In two instances the individual's cardiac output increased at one point during acceleration to values exceeding that of the control period prior to the exposure.

Three of the five subjects were unable to complete the 10 minute exposure

Table 8

Change in Cardiac Index from the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Headward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value L/min./m^2	Per Cent Change in Cardiac Index Duration of the Exposure (minutes):					Post-run (1 g)
		0 – 1	1 – 3	3 – 5	5 – 7	7 – 10	
2	(5) 3.8 [2.5 – 5.1]	(5) *–5 [–23 to +13]	(5) –5 [–20 to +13]	(5) –17 [–10 to –25]	(5) –7 [–15 to +4]	(5) –13 [–3 to –25]	(4) –3 [–8 to +3]
3	(5) 3.4 [2.5 – 4.2]	(5) –20 [–32 to –11]	(5) –6 [–19 to +19]	(5) –19 [–29 to –10]	(5) –16 [–31 to –4]	(4) –17 [–21 to –8]	(5) +4 [–24 to +28]
3½	(5) 3.2 [2.4 – 4.3]	(4) –17 [–25 to –4]	(5) –14 [–30 to +8]	(4) –7 [–35 to +27]	(4) –11 [–30 to +35]	(1) –28 –	(5) +12 [–3 to +22]
Control (1 g)	(4) 3.2 [2.4 – 4.1]	(4) –11 [–20 to –4]	(4) +1 [–6 to +7]	(4) +11 [0 to +22]	(4) –2 [–12 to +5]	(5) +1 [–3 to +5]	–

( ) Number of individual determinations.
[ ] Range of individual determinatons.
* Minus sign indicates a decrease during acceleration.

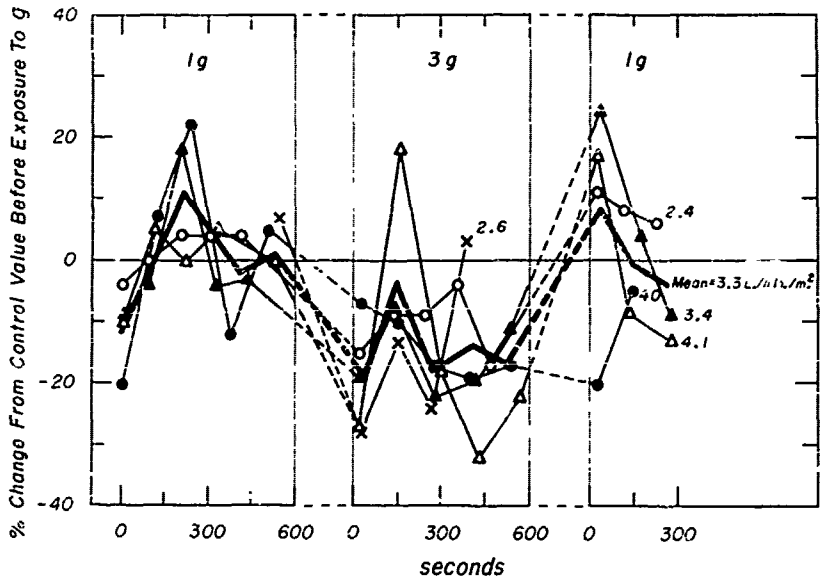

Figure 9. Variation in cardiac output prior to and during 10 minute exposures to 3 g headward acceleration in 5 healthy subjects.

Each symbol represents an individual subject's determination of flow at that particular time located on the abscissa. The heavy line drawn throughout the figure represents a mean value at any particular moment. The individual's average control value is shown at the end of each plot.

to $3\frac{1}{2}$ g. After approximately 5 minutes at $3\frac{1}{2}$ g. these subjects reported loss of peripheral vision; two of the three continued the exposure to the point where central vision was also lost. Upon termination of this exposure to $3\frac{1}{2}$ g, one subject experienced a period of bradycardia (rate 50-55), a 20 to 30 mm. Hg. fall in systolic arterial pressure, diffuse sweating, and near syncope. After 2-3 minutes with the head between the knees in a semi-crouch position the symptoms abated and the heart rate and blood pressure returned to normal levels. Figure 10 is a continuous plot of this subject's heart rate and aortic systolic and diastolic pressure prior to, during, and following his exposure to the $3\frac{1}{2}$ g acceleration for 520 seconds.

In an effort to elicit the cause for these unexpected reactions, all 5 subjects were exposed to the identical profiles of acceleration on another day, without the introduction of the intravascular catheters. ECG leads were taped to the chest to monitor any change in heart rate during the exposure and the subject's response to both peripheral and central light signals was recorded. No difficulties of the type described were encountered and all subjects completed the three exposures to 2, 3, and $3\frac{1}{2}$ g for 10 minutes, respectively, without visual symptoms, bradycardia, or syncope.

With a single exception, the heart rate systematically increased in response to headward acceleration, the degree of increase becoming greater with higher levels of acceleration (table 9). The average percent increase

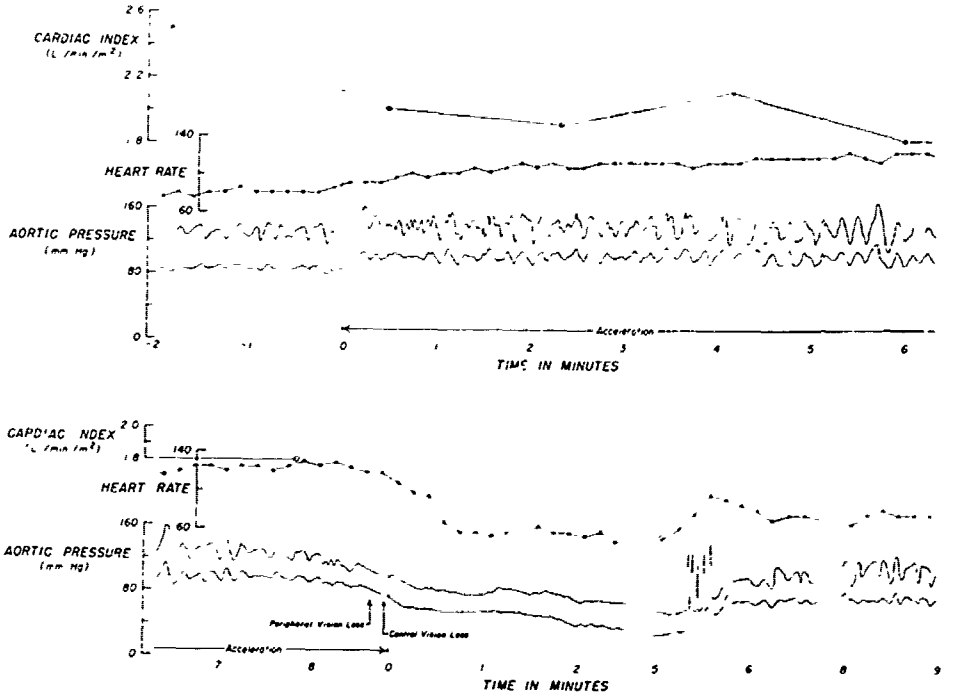

Figure 10. Plot of hemodynamic data during an $8\frac{1}{2}$ minute exposure to 3.5 g associated with development of a vasovagal type reaction in a normal subject (No. 4) occurring towards the end and following the termination of the exposure.

Note the large cyclic fluctuations in aortic pressure usually seen prior to and during the exposure. Following the onset of acceleration, the frequency of these variations in pressure increased. The intermittent determinations of cardiac output showed an initial decrease with the onset of acceleration, but no significant further systematic change as the exposure was prolonged to $8\frac{1}{2}$ minutes. During the 7th minute of the exposure the variation in aortic pressure decreased and there was an actual fall in both systolic and diastolic pressure beginning at minute 8. The heart rate failed to increase in response to this decrease in pressure and the subject experienced loss of peripheral and then central vision prior to termination of the exposure. The pressure and heart rate continued to fall following stoppage of the centrifuge until 5 minutes after termination of the exposure when the subject became nauseated to the point of retching. The pressure and heart rate then rose to normal values and the normally present cyclic fluctuations in aortic pressure returned approximately 8 minutes after the termination of the acceleration.

Table 9

Changes in Heart Rate from the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Headward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value beats/min.	Per cent change in Heart Rate					
		Duration of the Exposure (minutes):					
		0 - 1	1 - 3	3 - 5	5 - 7	7 - 10	Post-run (1 g)
2	(5) 82 [75 - 91]	(5) +15 [+5 to +24]	(5) +11 [+5 to +21]	(5) +12 [+4 to +26]	(5) +16 [+4 to +32]	(5) +15 [+4 to +35]	(4) +4 [-2 to +8]
3	(5) 81 [72 - 92]	(5) +16 [-4 to +35]*	(5) +25 [+13 to +39]	(5) +27 [+12 to +44]	(5) +32 [+14 to +50]	(4) +30 [+22 to +43]	(5) -3 [-15 to +8]
3½	(5) 79 [72 - 88]	(4) +36 [+18 to +54]	(5) +47 [+26 to +56]	(4) +51 [+27 to +73]	(4) +55 [+30 to +77]	(1) +72 [-]	(5) -2 [-13 to +11]
Control (1 g)	(4) 76 [72 - 79]	(4) 0 [-5 to +4]	(4) +1 [-9 to +10]	(4) -1 [-9 to +6]	(4) -4 [-10 to +2]	(5) +1 [-11 to +14]	

( ) Number of individual determinations.
[ ] Range of individual determinations.
* Minus sign indicates a decrease during acceleration.

when compared with the temporally contiguous control value was similar to that obtained in the first series of experiments and ranged from 11 to 16, from 16 to 32, and from 36 to 72 percent at 2, 3, and $3\frac{1}{2}$ g, respectively. As the acceleration was prolonged to 10 minutes, the average percent increase became greater during the 3 and $3\frac{1}{2}$ g exposures while the individual variation remained about the same. The average variation during the control (1 g) period was -4 to +1 percent of the individual's average control value.

As would be expected from previous comparisons, the average control values for stroke index were approximately the same as in the initial series of headward acceleration studies, (table 10). Following the initial decrease in stroke volume with the onset of acceleration, there was no systematic alteration in the average percent decrease throughout the 10 minute exposures to acceleration. The average decrease ranged from 13 to 24, from 23 to 36 and from 26 to 58 percent of the individual's control value prior to the exposure.

The control values for mean arterial pressure (table 11) were in close agreement with those of the previous series of studies. With the exception of three isolated instances, a consistent increase in mean arterial pressure was recorded in all 5 subjects upon exposure to acceleration. The average percent increase in mean arterial pressure from the individual temporally contiguous control value remained relatively stable during the 10 minute period of acceleration, ranging from 2 to 6, from 15 to 18, and from 4 to 21 percent at 2, 3, and $3\frac{1}{2}$ g, respectively. During the 10 minute control period, the mean arterial pressure remained within -8 to +5 percent of the individual's average control value.

The control values for total peripheral resistance also were relatively the same as those recorded in the initial series of experiments, (table 12) the average per cent variation during the 10 minute control period ranging from -9 to +11 percent of the individual's average control. On the average the total peripheral resistance increased with the onset of acceleration and the degree of this increase ranged from 11 to 27, from 24 to 47, and from 38 to 46 percent of the individual's control value prior to the exposure at 2, 3, and $3\frac{1}{2}$ g, respectively. No systematic increase or decrease could be determined as the acceleration was prolonged, the range of average values not exceeding those recorded during the control period.

Table 10

Changes in Stroke Index from the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Headward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value cc/stroke/m^2	Per cent change in Stroke Index					
		Duration of the Exposure (minutes):					
		0 - 1	1 - 3	3 - 5	5 - 7	7 - 10	Post-run (1 g)
2	(5) 47 [28 - 67]	(5) -18 * [-36 to -7]	(5) -13 [-36 to +8]	(5) -24 [-39 to -12]	(5) -19 [-34 to -3]	(5) -24 [-49 to -7]	(4) -3 [-13 to +3]
3	(5) 42 [30 - 58]	(5) -31 [-50 to -19]	(5) -23 [-43 to -4]	(5) -35 [-50 to -27]	(5) -36 [-45 to -29]	(4) -36 [-46 to -24]	(5) +10 [-9 to +27]
3½	(5) 41 [27 - 55]	(4) -26 [-51 to +10]	(5) -41 [-54 to -30]	(4) -36 [-64 to 0]	(4) -40 [-62 to -30]	(1) -58 [-]	(5) +18 [-4 to +40]
Control (1 g)	(4) 46 [32 - 56]	(4) -11 [-23 to -3]	(4) 0 [+6 to +14]	(4) +12 [-3 to +30]	(4) +1 [-5 to +7]	(5) 0 [-15 to +7]	-

( ) Number of individual determinations.
[ ] Range of individual determinations.
* Minus sign indicates a decrease during acceleration.

Table 11

Changes in Mean Aortic Pressure from the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Headward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value mm. Hg.	Per cent change in Mean Aortic Pressure — Duration of the Exposure (minutes): 0 - 1	1 - 3	3 - 5	5 - 7	7 - 10	Post-run (1 g)
2	(5) 94 [84 - 104]	(5) +6 [-3 to +12]*	(5) +6 [+3 to +9]	(5) +4 [-2 to +7]	(5) +2 [-6 to +7]	(5) +6 [+1 to +12]	(4) +1 [-4 to +7]
3	(5) 90 [83 - 94]	(5) +18 [+16 to +23]	(5) +17 [+12 to +22]	(5) +17 [+15 to +20]	(5) +17 [+11 to +19]	(4) +15 [+10 to +17]	(5) +1 [-10 to +6]
3½	(5) 92 [81 - 100]	(4) +16 [+9 to +25]	(5) +20 [+17 to +27]	(4) +21 [+11 to +27]	(4) +20 [+9 to +37]	(1) +4 [ ]	(4) -7 [-4 to -14]
Control (1 g)	(4) 90 [83 - 95]	(4) -1 [-3 to +1]	(4) +1 [-1 to +5]	(4) +1 [-4 to +3]	(4) 0 [-1 to +2]	(4) 0 [-8 to +4]	

( ) Number of individual determinations.
[ ] Range of individual determinations.
* Minus sign indicates a decrease during acceleration.

Table 12

Changes in Total Peripheral Resistance from the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Headward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value dyne sec. cm.$^{-5}$	Per cent change in Total Peripheral Resistance					
		Duration of the Exposure (minutes):					
		0 - 1	1 - 3	3 - 5	5 - 7	7 - 10	Post-run (1 g)
2	(5) 1090 [650 - 1540]	(5) +11 [-13 to +32]	(5) +16 [-2 to +34]	(5) +27 [+19 to +40]	(5) +11 [-1 to +26]	(5) +22 [+9 to +46]	(4) +3 [-9 to +14]
3	(5) 1150 [830 - 1550]	(5) +47 [+29 to +66]	(5) +24 [0 to +43]	(5) +44 [+28 to +56]	(5) +43 [+20 to +84]	(4) +40 [+30 to +49]	(5) 0 [-24 to +30]
3½	(5) 1230 [770 - 1570]	(4) +41 [+18 to +79]	(5) +44 [+18 to +82]	(4) +38 [0 to +91]	(4) +46 [-14 to +110]	(1) +46 [-]	(5) -18 [0 to -26]
Control (1 g)	(4) 1120 [800 to 1620]	(4) +11 [+6 to +20]	(4) 0 [-7 to +4]	(4) -9 [-17 to -1]	(4) +2 [-3 to +10]	(5) 0 [-5 to +8]	

( ) Number of individual determinations.
[ ] Range of individual determinations.
A minus sign indicates a decrease during acceleration.

III. The Effects of Forward Acceleration of 10 Minutes Duration

For this series of accelerations the subject was supported in a supine, seated position by a nylon net strung tightly to a contoured metal frame (figure 11). The trunk formed a 12 degree angle with the floor of the centrifuge cockpit and the legs were flexed to form 100 degree angles at both the hips and knees. For each determination of cardiac output during these forward acceleration studies, the turning of stopcocks for blood withdrawal, indicator injection, and flushing of the catheter systems was performed by the subject. The activation of the automatic withdrawal and injection syringes as well as the starting and stopping of the centrifuge were under the control of the center observer. No overhead observer was necessary during these experiments.

Three subjects (No. 3, 4, and 6 of the original panel) were exposed to 3 separate plateau levels of forward acceleration of up to 10 minutes duration in sequence at 2, $3\frac{1}{2}$, and 5 g, respectively. The sequence of exposures was reversed in a repeat experiment with subject No. 4 as well as in an experiment with subject No. 7. In addition, subjects No. 1 and 5 were exposed to two separate plateau levels of 2 and $3\frac{1}{2}$ g for up to 10 minutes duration. A total of 29, 30, and 21 dye dilution curves were recorded during these exposures to 2, $3\frac{1}{2}$, and 5 g, respectively. Thirty-five control (1 g) determinations of cardiac output were performed prior to and following the 80 determinations carried out during centrifuge rotation. In addition to these controls 5 of the 6 subjects' cardiac outputs were determined 5 separate times in rapid succession during a 10 minute period with the centrifuge stationary. The catheterization procedure was identical to the two previous series of experiments. All pressures were referenced to the mid chest at the level of the 3rd intercostal space at the sternum. The shi in reference level was recorded by means of the thistle-tube system described previously. Mechanical extensions were attached to the two-way stopcocks on the radial cuvette and venous catheter systems and brought to a central control panel which the subject operated as directed over the intercommunication system by an observer reading from a printed sequence (figure 12).

The results are shown in Tables 13 to 17 and Figures 13 to 17, which depict the effects of forward acceleration of up to 10 minutes duration on cardiac output, heart rate, stroke volume, mean arterial pressure, and total peripheral resistance. As will be evident from the results depicted graphically, a significant difference occurred in the values obtained during the 2 and 5 g exposures respectively when the sequence of exposures to acceleration was reversed. The results of the two experiments in which the sequence was reversed, therefore, were omitted from the analysis of per cent changes in relation to duration of exposure in tables 13 through 17.

The average and range of control values for cardiac output in the seated, supine position (table 13) are significantly higher than those obtained in the upright, seated position prior to the headward acceleration studies. The average cardiac output varied from -3 to +9 percent of the average control value over a 10 minute period of time, and the individual variation was from -10 to +13 percent of the individual's average control. This degree of variation was significantly less than that associated with the upright, seated position. With the onset of forward acceleration, the cardiac output

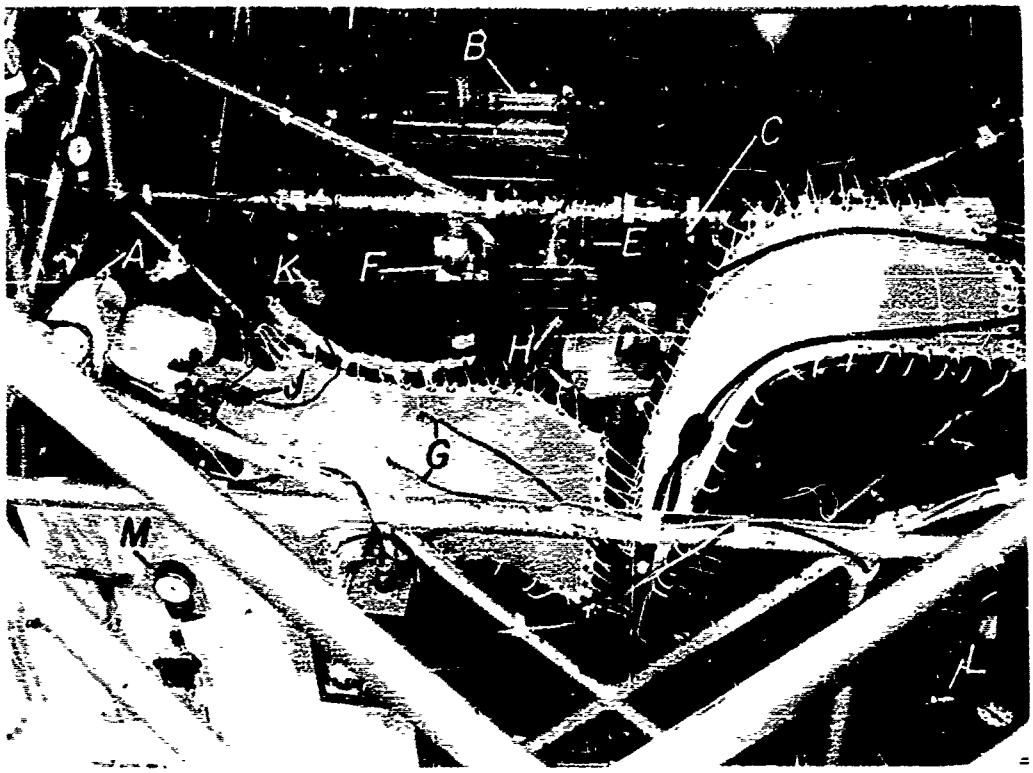

Figure 11. Contoured nylon support system used for the forward acceleration studies. A.) Plaster headpiece worn by subject to permit firm attachment of the earpieces to measure blood oxygen saturation and ear opacity pulse. B.) Automatic syringe for blood withdrawal and infusion from the radial artery through the cuvette oximeter C.). D.) Arm and wrist support to maintain position of left arm and wrist containing arterial and venous catheters. E.) Strain gauges for measurement of aortic, radial artery, and right atrial pressures. F.) Thistle tube and connecting tubing which when filled with fluid and attached to subject were opened to the manometer systems during exposures to the levels of acceleration studied to allow accurate recording of shifts in baseline imposed on these systems by the acceleration and the shift of the subject in the seat. G.) Electrocardiograph leads. H.) Panel with 5 control knobs attached to stopcocks which permit the subject to flush the aortic catheter, interchangeably connect both the right atrial catheter with either the manometer or the dye injection syringe and the radial artery needle with either the manometer or the automatic withdrawal infusion syringe, and allow adequate flushing of the syringe tubing to prevent clotting between determinations of cardiac output. The mechanism for flushing the radial artery catheter which was activated by the left foot is not shown. J.) Oral airway containing thermocouple for recording of respiration and K.) mouth microphone for intercommunication system. L.) Automatic dye injection syringe. M.) Part of pressurized flushing system to keep catheter-manometer systems from clotting.

Table 13

Changes in Cardiac Index From the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Forward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value L/min/m²	Per Cent Change in Cardiac Index — Duration of the Exposure (minutes): 0 – 1	1 – 3	3 – 5	5 – 7	7 – 10	Post-run (1 g)
2	(4) 5.4 [4.0 – 7.4]	(4) −18 [−32 to −2]	(4) −12 [−27 to −8]	(4) −19 [−29 to −8]	(4) −16 [−36 to −5]	(3) −16 [−35 to −7]	(3) −28 [−39 to −22]
3½	(5) 3.9 [2.6 to 4.9]	(5) +16 [0 to +34]	(5) +21 [+8 to +58]	(4) +26 [+4 to +58]	(4) +20 [+4 to +34]	(3) +22 [+8 to +22]	(5) +20 [+6 to +38]
5	(4) 4.0 [3.3 to 5.4]	(4) +11 [−8 to +26]	(4) +15 [−3 to +28]	(2) +34 [−24 to +44]	(1) +24 [–]	–	(3) +15 [+2 to +32]
1	(3) 3.9 [3.1 to 5.1]	(3) −2 [−6 to 0]	(3) −1 [−2 to 0]	(3) −3 [−10 to +3]	(3) +9 [+6 to +13]	(3) +2 [−6 to +6]	

( ) Number of individual determinations.
[ ] Range of individual determinations.
* A minus sign indicates a decrease during the period of acceleration.

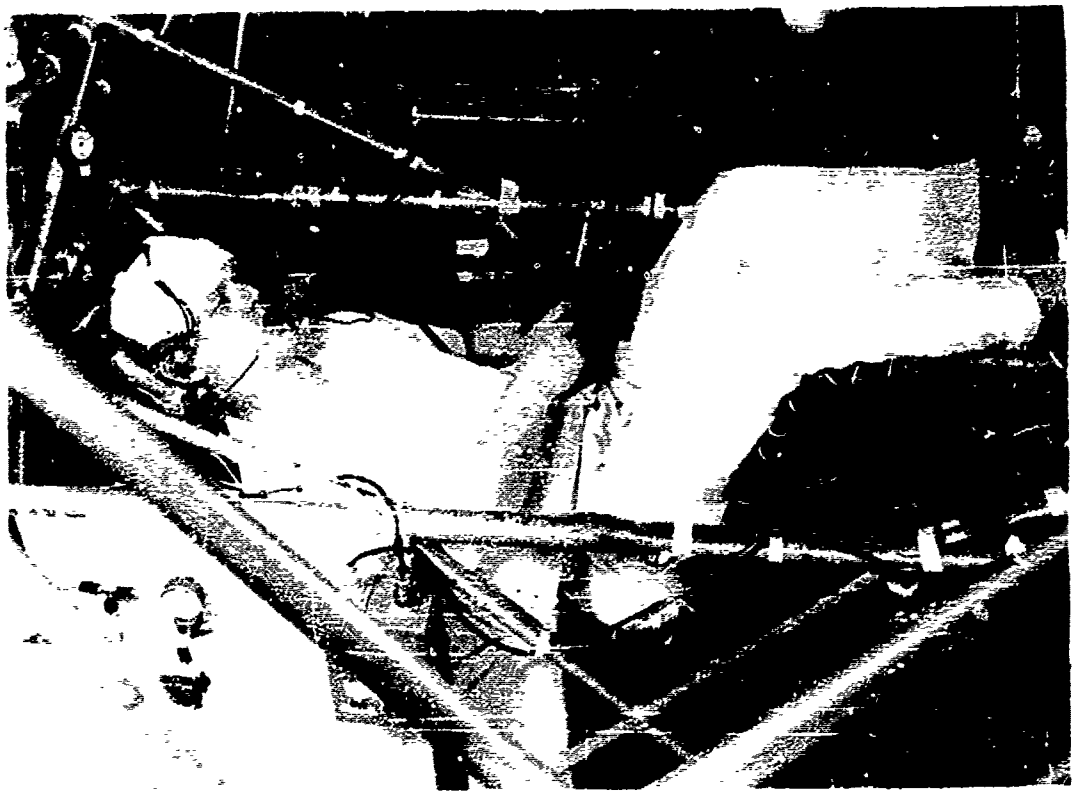

Figure 12. Subject shown operating the remote stopcock controls from a central control panel. The sequence of operation is received through the earphones from an observer stationed outside the centrifuge room. Subject is able to communicate with the observer by means of the microphone shown just above the mouth piece. The oximeter for recording the blood content of the ear and the oxygen saturation of this blood is shown in place and fixed in position on the plastic helmet.

decreased from control levels in every case but one during the 2 g exposure (figure 13) and increased from control levels in all but two instances during the 3½ and 5 g exposures. As these exposures were prolonged to 10 minutes, multiple determinations of output at approximately 2 minute intervals did not demonstrate any systematic changes in the average values, the average variation not exceeding that of the control period. The average decrease in cardiac output ranged from 12 to 19 percent of the temporally contiguous control values during the 2 g exposure. The average increase in output ranged from 16 to 26 and from 11 to 34 percent of the control value prior to exposures to 3½ and 5 g, respectively. Since the control value immediately following the 2 g acceleration in those subjects exposed in the original

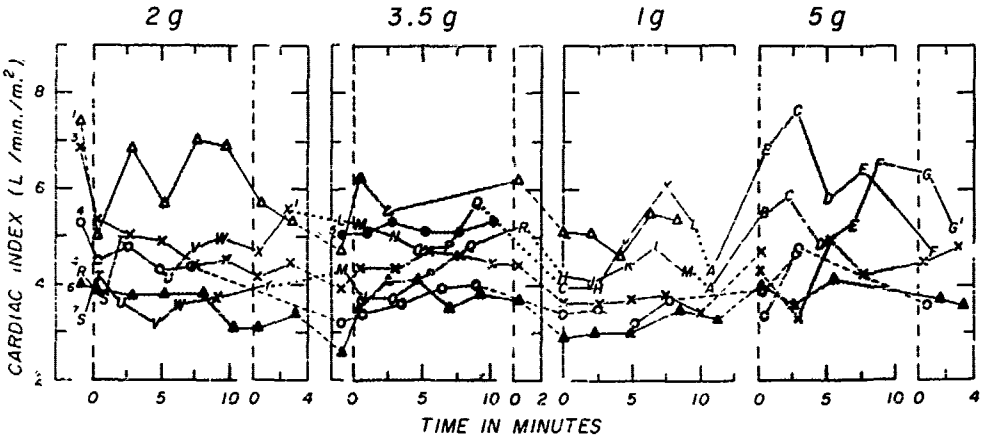

Figure 13. Effect of forward acceleration on cardiac output in six healthy subjects.

Subjects 1, 3, 4, 5, and 6 are represented by symbols (for key, see table 1), and every determination of cardiac output prior, during and following exposures to 2, $3\frac{1}{2}$, and 5 g forward accelerations respectively are plotted in relation to time shown on the abscissa. In addition, subject 7 and subject 4 were exposed to the same three levels of acceleration in a reverse sequence (5, $3\frac{1}{2}$, and 2 g) and the determinations performed during these experiments are plotted using letters of the alphabet in the sequence in which they were performed. Those determinations joined by the solid lines indicate no interruption in the time sequence. Those values joined by the dashed lines indicate an indeterminate time interval between two determinations. Dotted lines were used to allow the reader to follow the determinations of the two subjects in whom the sequence of exposure was reversed.

With the onset of 2 g acceleration, subjects 1, 3, 4, and 6 showed a decrease in output but as the exposure was prolonged to 10 minutes, multiple determinations of output at approximately two minute intervals failed to demonstrate any systematic change. It is probable that the first control determination of output was associated with some degree of anxiety on the part of these subjects associated with the start of what they knew would be a grueling experience. The control 1 g values immediately following the 2 g exposure showed an even greater decrease in output in relation to the initial control value possibly due to the decrease in anxiety associated with the successful negotiation of the first period of centrifuge rotation. In general the control levels of cardiac output decreased as the experiment progressed. This was also demonstrable in the two subjects whose exposures to acceleration were carried out in reverse sequence.

sequence shows an even greater decrease in output, it is possible that the first control determination was associated with a degree of anxiety on the part of these subjects prior to the first dye injection, and exposure to acceleration in this position. It is of interest in this regard that the control level of cardiac output decreased as the experiment progressed (table 13, figure 13).

Two of the five subjects were unable to complete the 5 g acceleration; one because of nausea and vomiting and the other because of substern 1 chest pain. No one reported any visual symptoms nor were there any vasovagal episodes as occurred with the prolonged headward accelerations.

There was a decrease in the average heart rate (table 14, figure 14) ranging from 4 to 16 percent of the control value prior to acceleration during the exposure to 2 g. The heart rate increased from the temporally contiguous

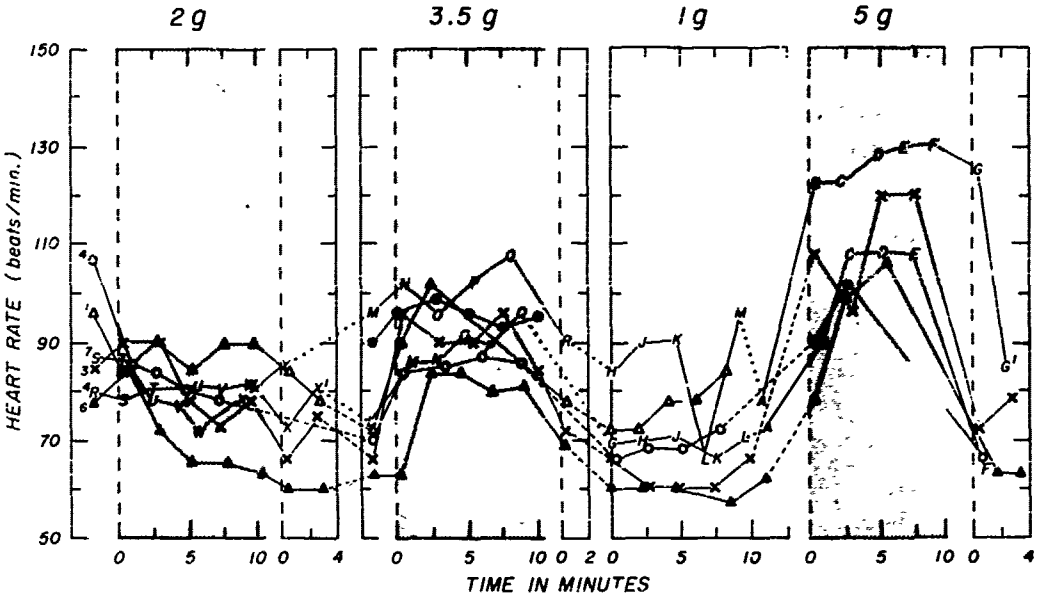

Figure 14. Effect of forward acceleration on heart rate in six healthy subjects.

See legend figure 13, for explanation of the symbols. Measurements of heart rate were made from that portion of the record obtained simultaneously with the determination of cardiac output. In response to acceleration, the heart rate increased during the $3\frac{1}{2}$ and 5 g exposures but varied at the 2 g level.

control value in every case during the $3\frac{1}{2}$ and 5 g exposures (table 14). The average and range of increase was 19 to 29 and from 35 to 82 percent at the $3\frac{1}{2}$ and 5 g levels, respectively. The average variation during the 10 minute control period was -1 to +6 percent of the individual's average control value.

Table 14

Changes in Heart Rate from the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Forward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value beats/min.	Per cent changes in Heart Rate Duration of Exposure (Minutes): 0 - 1	1 - 3	3 - 5	5 - 7	7 - 10	Post-run
2	(4) 91 [78 - 107]	(4) -4 [-20 to +8]	(4) -7 [-21 to +7]	(4) -15 [-25 to -7]	(4) -16 [-27 to -6]	(3) -11 [-19 to -6]	(3) -19 [-23 to -12]
3½	(5) 72 [63 - 90]	(5) +19 [0 to +45]	(5) +29 [+10 to +42]	(4) +25 [+7 to +36]	(4) +23 [+3 to +45]	(3) +21 [+7 to +28]	(5) +2 [-13 to +10]
5	(4) 71 [62 to 84]	(4) +35 [+25 to +64]	(4) +48 [+42 to +60]	(2) +77 [+71 - +82]	(1) +82 [-]	-	(3) +1 [-8 to +9]
1	(3) 66 [60 - 77]	(3) 0 [-6 to +6]	(3) -1 [-6 to 0]	(3) -1 [-3 to +1]	(3) -2 [-5 to +1]	(3) +6 [+3 to +9]	

( ) Number of individual determinations.
[ ] Range of individual determinations.
A minus sign indicates a decrease during the period of acceleration.

In general the control values for heart rate in this position were lower than those recorded in the upright position in the previous studies of headward acceleration. The heart rate was higher during the first control determination of cardiac output prior to the exposure to 2 g than for any of the other control values for heart rate, thus supporting the interpretation stated in the prior paragraph that the subject was in a somewhat more excited state at this time than during the subsequent periods of the experiment.

The control values for stroke volume were significantly greater (table 15) in this position than those recorded for the upright seated position, as would be expected from previous comparisons. The change in stroke volume which took place with the onset of acceleration was extremely variable (figure 15) and on the average ranged from -20 to -4 and from -5 to +1 percent of the temporally contiguous control values during the 2 and $3\frac{1}{2}$ g exposures. There was a significant decrease in stroke volume of from -20 to

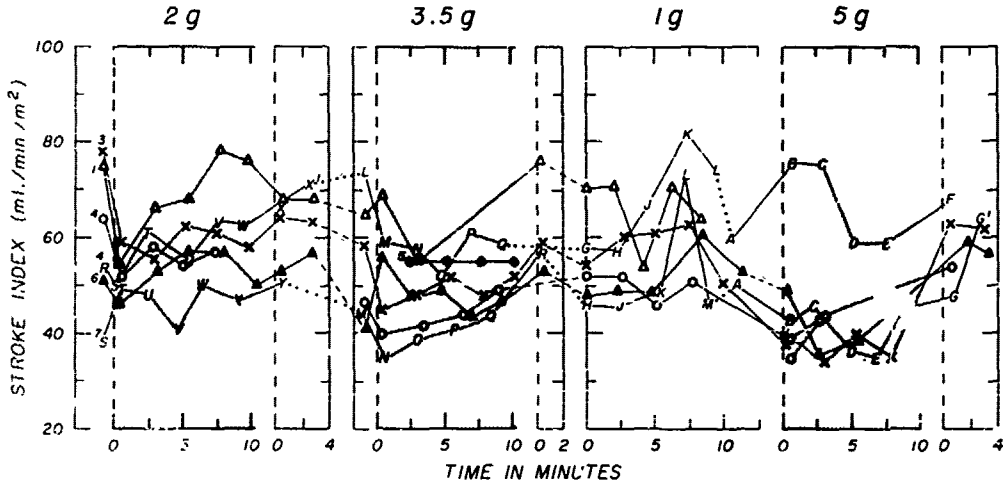

Figure 15. Effect of forward acceleration on stroke index in six healthy subjects. No striking systematic changes are evident.

-30 percent during the 5 g exposure to forward acceleration of 10 minutes duration. Following the determinations made during the first minute of the exposures, there was no systematic alteration in the values obtained during the subsequent 10 minutes of the exposure to acceleration. The average stroke volume ranged from -6 to +11 percent of the individual's control value during the 10 minute period of determinations with the centrifuge stationary.

Table 15

Changes in Stroke Index from the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Forward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value cc/stroke/m^2	Per cent change in stroke index					
		Duration of the Exposure (Minutes):					
		0 -1	1 - 3	3 -5	5 - 7	7 - 10	Post-run
2	(4) 67 [51 - 78]	(4) -20 * [-28 to -10]	(4) -10 [-29 to +4]	(4) -8 [-20 to +12]	(4) -4 [-22 to +12]	(3) -9 [-26 to +1]	(3) -8 [-18 to +2]
3½	(5) 53 [41 - 55]	(5) -1 [-24 to +36]	(5) -5 [-18 to +17]	(4) +1 [-12 to +19]	(4) +1 [-18 to +7]	(3) +1 [-12 to +15]	(5) +18 [0 to +28]
5	(4) 55 [51 to 64]	(4) -20 [-31 to -7]	(4) -24 [-14 to -33]	(2) -24 [-26 to -22]	(1) -31 -	-	(3) +14 [+6 to +24]
1	(3) 59 [52 - 66]	(3) -2 [-8 to +3]	(3) +2 [-6 to +8]	(3) -6 [-18 to +5]	(3) +11 [+8 to +17]	(3) -4 [-12 to +2]	

( ) Number of individual determinations.
[ ] Range of individual determinations.
* A minus sign indicates a decrease during the period of acceleration.

The control values for mean aortic pressure in the supine seated position were significantly higher than those recorded in the upright, seated position (table 16). A consistent increase in mean arterial pressure was recorded in all 5 subjects upon exposure to forward acceleration (figure 16).

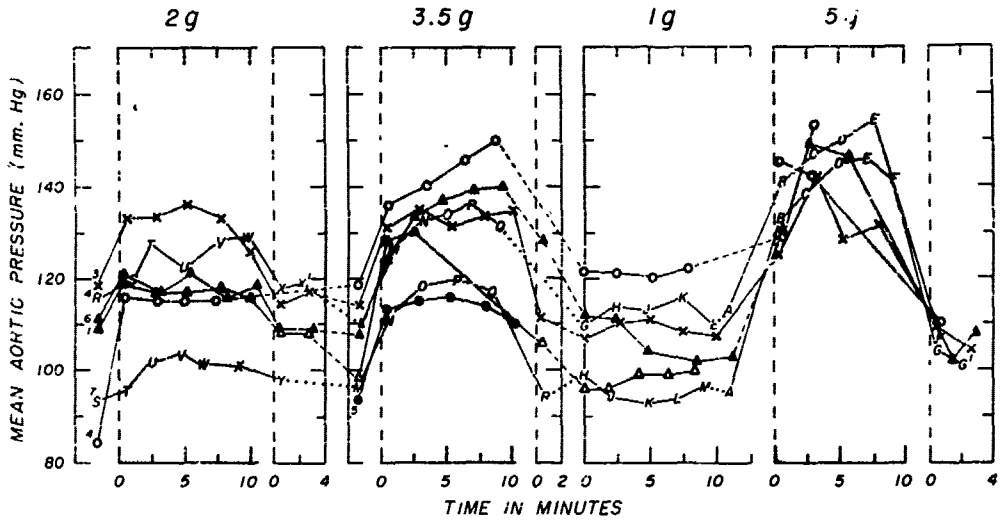

Figure 16. Effect of forward acceleration on aortic pressure in six healthy subjects. Note the increasing degree of systemic hypertension associated with exposures to higher levels of forward acceleration.

When compared with the control value obtained just prior to the exposure, the average increase ranged from 7 to 17, from 19 to 23, and from 17 to 31 percent at 2, $3\frac{1}{2}$, and 5 g, respectively. During the 10 minute control period, the individual's mean arterial pressure ranged from -4 to +5 percent of the average control value.

The control values for total peripheral vascular resistance (pressure/flow ratio) were approximately the same as those obtained in the previous 2 series of experiments (table 17). The average percent variation was from -5 to +5 percent of the individual's average control value, during the 10-minute control period when 5 successive determinations of cardiac output were carried out, when the centrifuge was stationary. When compared to the first control (1 g) value the average vascular resistance increased with the onset of the first exposure to acceleration of 2 g in those subjects exposed to the original sequence (figure 17). The average increase ranged from 29 to 33 percent during this first 10 minute exposure to acceleration in the experimental sequence. However, the average value obtained during the control (1 g) determination immediately following this exposure was

Table 16

Changes in Mean Aortic Pressure from the Temporally Contiguous Control Values at Various Periods During 10 Minute Exposures to Plateau Levels of Forward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value mm. Hg.	Per cent change in Mean Aortic Pressure					
		Duration of the Exposure (Minutes):					
		0 - 1	1 - 3	3 - 5	5 - 7	7 - 10	Post-run
2	(4) 106 [84 - 118]	(4) +17 [+9 to +38]	(4) +16 [+6 to +37]	(4) +17 [+7 to +37]	(4) +15 [+4 to +37]	(3) +7 [+6 to +9]	(3) -2 [-3 to -1]
3½	(5) 107 [95 - 118]	(5) +19 [+14 to +31]	(5) +23 [+18 to +33]	(4) +22 [+15 to +27]	(4) +23 [+16 to +29]	(3) +21 [+17 to +30]	(5) +3 [-4 to +9]
5	(4) 108 [100 - 122]	(4) +17 [+6 to +26]	(4) +29 [+16 to +44]	(2) +31 [+20 to +42]	(1) +22 -	-	(3) -3 [-10 to +2]
1	(3) 104 [98 - 109]	(3) +1 [-2 to +6]*	(3) +1 [-2 to +5]	(3) 0 [-2 to +2]	(3) -1 [-4 to +1]	(3) -1 [-3 to +2]	-

( ) Number of individual determinations.
[ ] Range of individual determinations.
* A minus sign indicates a decrease during the period of acceleration.

Table 17

Chang s in Total Peripheral Resistance from the Temporally Contiguous Control Values at Various Per'ods During 10 Minute Exposures to Plateau Levels of Forward Acceleration
(average values, 5 subjects)

Acceleration "g"	Control (1 g) value dyne sec. cm.$^{-5}$	Per cent change in Total Peripheral Resistance — Duration of the Exposure (Minutes): 0 - 1	1 - 3	3 - 5	5 - 7	7 - 10	Post-run
2	(4) 815 [610 - 1170]	(4) +36 [+12 to +57]	(4) +29 [+9 to +58]	(4) +37 [+13 to +65]	(4) +32 [+9 to +76]	(3) +38 [+11 to +64]	(3) +37 [+24 to +59]
3½	(5) 1140 [750 - 1500]	(5) +6 [-1 to +19]*	(5) +6 [-7 to +13]	(4) +2 [-5 to +17]	(4) +2 [-5 to +14]	(3) +6 [+4 to +8]	(5) -11 [-21 to -4]
5	(4) 1150 [750 - 1340]	(4) +10 [-8 to +40]	(4) +18 [-4 to +37]	(2) 0 [-16 to +15]	(1) 0 -		(3) -17 [-30 to -9]
1	(3) 1120 [770 - 1430]	(3) +4 [-1 to +14]	(3) +3 [-1 to +9]	(3) +5 [0 to +12]	(3) -5 [-15 to +5]	(3) -3 [-10 to +4]	

( ) Number of individual determinations.
[ ] Range of individual determinations.
* A minus sign indicates a decrease during the period of accelerat on.

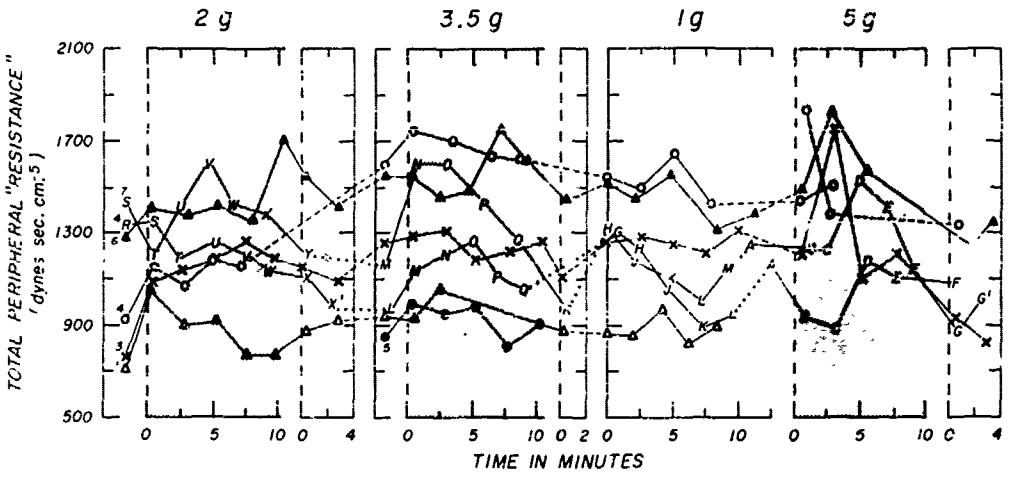

Figure 17. Effect of forward acceleration on total peripheral "resistance" in six healthy subjects. No striking systematic changes are evident.

significantly higher than the first 1 g value, and did not differ significantly from the values obtained during this exposure. This suggests as did the values for cardiac output and heart rate, that the hemodynamic status of these subjects was significantly different during the first control determination of cardiac output carried out at the onset of the experiment prior to any exposure to acceleration than during any of the subsequent control determinations carried out during the remainder of the experiment. There was no systematic alteration in resistance as the exposure to acceleration was prolonged. The peripheral vascular resistance tended to be increased slightly at $3\frac{1}{2}$ and 5 g in relation to the temporally contiguous control value prior to the exposures. There was a systematic decrease in vascular resistance of 11 and 17 percent during the determinations at 1 g immediately following these exposures.

Forward acceleration produced a striking effect on right atrial pressure in these subjects as shown in figure 18. This increment in atrial pressure became greater as the magnitude of acceleration increased, a four-fold increase taking place in some cases at 5 g. Upon reaching the plateau acceleration there was a gradual but significant decline in this rise in pressure attained at the onset of acceleration as the exposure was prolonged to 10 minutes. In relation to the value obtained at 1 g prior to each exposure, there is a systematic decrease in right atrial pressure at 1 g immediately after each exposure, suggesting a loss of circulating blood volume during the exposure and an increase in the capacity of the vascular bed.

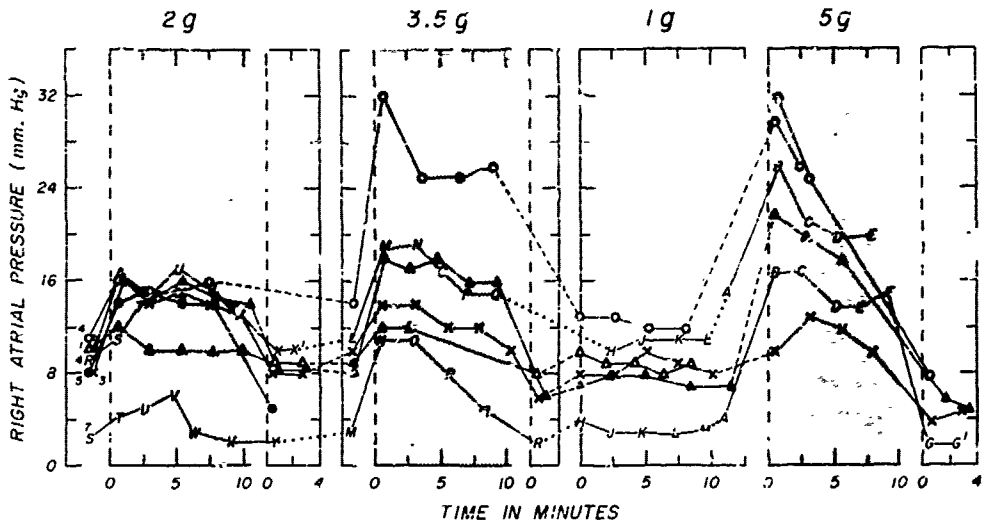

Figure 18. Effect of forward acceleration on right atrial pressure in six healthy subjects. Note: (1) the increasing magnitude of the increment in atrial pressure with increased levels of acceleration; (2) the progressive decrease in right atrial pressure from the maximum level attained at the onset of acceleration; and (3) that in relation to the value at 1 g before each exposure there is a systematic decrease in right atrial pressure at 1 g immediately after each exposure. This suggests loss of circulating blood volume during the exposure and/or an increased capacity of the vascular bed.

As was the case in the headward acceleration studies, marked decreases in arterial blood oxygen saturation took place during exposure to forward acceleration. The changes taking place in subject 7 during exposure to 5 g acceleration are shown in figure 19. With the subject breathing air, an immediate fall in arterial blood oxygen saturation was recorded via the cuvette oximeter upon exposure to acceleration. This rapid decrease in saturation with the onset of acceleration is compatible with the occurrence of a "physiologic" right-to-left shunt. Upon termination of the exposure there was a slow return of oxygen saturation to normal levels. One interpretation of this phenomenon would be that a partial collapse of the dependent portions of the lungs may occur during the exposure, and would require some length of time to return to normal function. This same subject was exposed to 5 g for 30 seconds while breathing 99% oxygen. No decrease in arterial blood oxygen saturation took place within this length of time which is further evidence of a defect in the ventilation-perfusion function of the lung during forward acceleration.

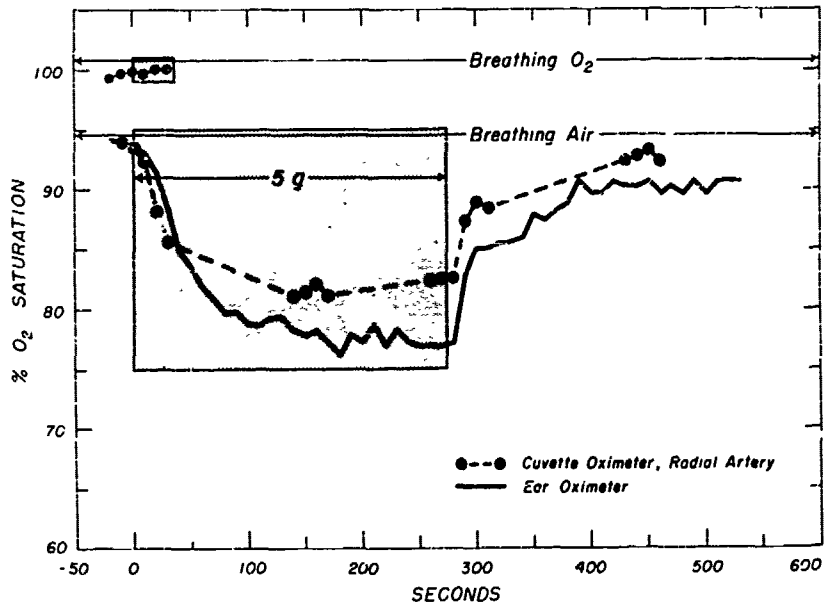

Figure 19. Changes in arterial oxygen saturation during exposure to a forward acceleration of 5 g when breathing air and oxygen (subject No. 7). Note: (1) that no decrease in arterial oxygen saturation occurred during a 30 second exposure when breathing 99.6% oxygen; (2) the rapidity of the decrease in saturation at the onset of the acceleration which is compatible with the occurrence of a "physiologic" right-to-left shunt and (3) the slowness of recovery following termination of the exposure which is compatible with the interpretation that partial collapse of the dependent portion of the lungs may occur during the exposure.

The measurement of cardiac output from dye-dilution curves recorded continuously by the cuvette oximeter is an accepted procedure (ref. 12). Satisfactory agreement between the indicator-dilution and the direct Fick methods under usual laboratory conditions has been established by studies from many laboratories (ref. 6). It must be recognized, however, that under the extreme conditions of these studies disturbances in the circulation such as retention of dye in dependent portions of the lung, where blood may tend to stagnate or flow more slowly, may invalidate the method. There is no direct evidence from these experiments that dye was not lost in this manner, or that other disturbances in the circulation may not have interfered with this method. But conversely, gross alterations in the time components of the dilution curves recorded in these experiments -- the appearance time, buildup time, passage time, and recirculation time of the dye -- as well as decreases in the disappearance slope of the curve might be expected if this were the case. Analysis of the time components of the dilution curves recorded during centrifugation in the upright seated position showed an average increase of 20 percent or less as compared with the curves measured during control conditions. The disappearance slope showed an average decrease of 20 percent during acceleration. These changes in the different time components of individual curves were similar, so that the contours of the curves recorded during acceleration were not apparently different from normal (figure 3). The generalized increase in time components and decrease in disappearance slope were consistent with the changes which would be expected with the decreases in cardiac output that were obtained in this series of studies of headward acceleration. It does not appear that the changes in contour of the curves are of such nature or magnitude as to invalidate the indicator-dilution method of determining cardiac output under any of these conditions.

A basic assumption of the indicator-dilution method is that the blood flow remains constant during the period of inscription of the curve (ref. 11). Accordingly, no attempt was made to determine cardiac output by this method during the initial 10 to 15 seconds of exposure to headward acceleration, for it is well known that dramatic cardiovascular compensatory reactions occur during this period (ref. 17). Recordings of blood pressure and other parameters suggest that the initial cardiovascular compensatory reactions are largely completed within the first 15 seconds of the exposure (ref. 17). Hence it might be anticipated that the blood flow during the period 20 to 40 seconds after the onset of such an exposure would be sufficiently stable to allow a determination of cardiac output by the indicator-dilution method during this time. In any event, this has been a necessary assumption in these studies.

The use of indocyanine dye permitted multiple determinations of cardiac output without systemic toxicity or discoloration of the subject's skin. Also, the recording of dye concentration in whole blood was unaffected by changes in oxygen saturation (ref. 2). The latter consideration was important because of the variations (87 to 99 percent) in arterial blood

oxygen saturation which occurred during the exposures to headward acceleration and those of even greater magnitude that occurred during forward acceleration.

To refer all measurements of pressure to the third intercostal space at the sternum or to the mid chest may seem arbitrary and to some degree meaningless from the hemodynamic viewpoint if the heart moves downward or backward under the influence of acceleration. Roentgenograms of the upper part of the body taken at the end of expiration in subjects experiencing headward accelerations to 5 g (ref. 13) revealed less than 1 cm. of shift in the base of the heart when the distance between the base of the skull and the juncture of the right auricle with the aorta was measured. Thus it is possible to reference pressures recorded during headward acceleration to an external point on the body (in this case, the third intercostal space at the sternum) and still be certain that any shift of this point will be reflected by a shift in heart level. On the other hand, the heart has been shown to shift posteriorly as much as 2 cm. during forward acceleration up to 6 g. Since the external reference point for pressures recorded during forward acceleration was located at the mid chest position at the level of the third intercostal space, it was not possible to take into account any change in heart level which might occur within the chest upon exposure to acceleration. As shown in table 16 (figure 16), the arterial pressure recorded during forward acceleration increased. Any posterior shift of the heart which might take place during acceleration would act to decrease the level of pressure being recorded. These results then must be considered in light of these facts.

Following the initial 15 seconds of headward acceleration at 4 g, all subjects in the first series of experiments reported clear vision. Further validation of the measurements of aortic pressure was gained by calculating the intra-arterial pressure at heart level necessary to maintain adequate arterial pressure at eye level to prevent interruption of the circulation to the eyes and the resulting loss of vision (blackout). Since it has been shown that 20 mm. Hg. of pressure at eye level is necessary to prevent collapse of the retinal vessels as a result of intra-ocular pressure (ref. 10), and since the distance from the eye to the third intercostal space at the sternum measured 30 to 35 cm. in these subjects, it was calculated that a mean pressure of 112 to 128 mm. Hg. at heart level was necessary to prevent blackout at 4 g. All 6 subjects had pressures within or above this range during the 20 to 40 second period of the exposure during which these particular measurements were made.

The hydrostatic pressure differences between the upper and the dependent portions of the body in the seated position, which are greatly exaggerated during exposure to acceleration, render the interpretation of the physiologic significance of values for pressure/flow ratios calculated under these circumstances even more difficult than usual. These estimated values which are referred to heart level have no direct significance regarding the relationship between blood pressure and flow through segments of the circulation at different levels above and below the heart. The pressure/flow ratio as calculated does, however, have some significance in relation to the resistance against which the left side of the heart was pumping under the conditions studied.

The relatively small degree of protection afforded by the rather ineffectual and uneven pressurization of the lower body, provided by inflation of the G-3A suit during headward acceleration, did not give a good basis upon which to investigate the mechanism of such protection. Furthermore, the protection afforded by such devices is more obvious during the initial 15 seconds of exposure to acceleration than during the subsequent period, when the immediate cardiovascular compensatory reactions have had sufficient time to come into full effect. The mean aortic pressure was the only parameter studied in which a statistically significant difference was found between th values obtained during exposure to headward acceleration with and without inflation of the anti-blackout suit. Since a systematically smaller decrease in cardiac output was not demonstrated during these exposures with the suit inflated, it appears that the mechanism for the increase in aortic pressure produced by the suit was an increase in the level of systemic arterial resistance. The average increase in pressure/flow ratio obtained during exposures with the suit inflated was in fact greater than for the exposures without suit protection. This difference, however, did not attain statistical significance (table 6). It has been demonstrated previously that inflation of an anti-blackout suit elevates the base of the heart 2 to 3 cm. (ref. 13). If under this circumstance blood pressure were unchanged at true heart level, such an effect would produce an increase in blood pressure to 2 to 3 mm. Hg. per g when measured at a fixed external point on the body such as the level of the third interspace at the sternum. Theoretically this effect would provide a protection of less than 0.5 g at an acceleration of 4 g.

These considerations lend support to the concept that the protection afforded by an anti-blackout suit is related to the degree of hypertension at heart level which inflation of the suit produces during exposure to acceleration (ref. 16). The results of the present study suggest that this increase in arterial pressure results chiefly from an increase in systemic vascular resistance rather than from an increase in cardiac output.

It should be re-emphasized that the determinations of cardiac output during the 60 second exposures to headward acceleration in the first series of studies were limited to the period from 20 to 40 seconds after the onset of plateau levels of acceleration. The changes in cardiac output demonstrated during this period of an exposure to acceleration cannot be extrapolated with validity to other periods during the exposure, as demonstrated by the results of the multiple determinations of flow performed during the 10 minute exposures to headward acceleration. Particularly this is true for the initial 10 to 15 seconds of exposure to accelerations with a rapid onset which at levels of greater than 3 g produce temporary insufficiency of circulation to the head and hence dramatic cardiovascular compensatory reactions presumably including changes in blood flow.

Elucidation of the changes in cardiac output and the relative distribution of this flow to the body in human subjects during the initial period of circulatory failure and cardiovascular compensation induced by headward acceleration requires the application of methods capable of determining instant-to-instant changes in blood flow in the intact organism. Neither the conventional direct Fick or indicator-dilution techniques using sudden single injections is applicable to these requirements. The method for continuous measurement of lower body flow in man described by Grace and co-workers (ref. 5) provides a possibility for such studies if the technical difficulties of its application during rotation at high speed on a centrifuge can be overcome.

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